A Dissertation on

CLINICAL PROFILE AND ANALYSIS OF RISK FACTORS OF MYOCARDIAL INFARCTION IN THE YOUNG (≤ 40 YRS)

(A CASE-CONTROL STUDY)

Dissertation submitted to

THE TAMIL NADU DR.M.G.R.MEDICAL UNIVERSITY CHENNAI.

with fulfillment of the regulations for the award of the degree of

M.D. (General Medicine)
Branch - I

KILPAUK MEDICAL COLLEGE,
CHENNAI.

SEPTEMBER 2006

CERTIFICATE

This is to certify that this dissertation on "**A STUDY OF CLINICAL PROFILE AND ANALSYIS OF RISK FACTORS OF MYOCARDIAL INFARCTION IN THE YOUNG (≤ 40 YRS)"** is a work done by **Dr.V.ARUMUGAM,** under my guidance during the period of 2003 - 2006. This has been submitted in partial fulfillment of the award of M.D. Degree in General Medicine (Branch-I) by the Tamil Nadu Dr.M.G.R. Medical University, Chennai.

Prof.Dr.K.S.SAI KUMAR, M.D.,
Professor and Head of Department
Department of Medicine
Kilpauk Medical College & Hospital,
Chennai.

Dr.G.RAJENDRAN, M.D.,
Reader in Medicine,
Government Royapettah Hospital,
Kilpauk Medical College
Chennai.

THE DEAN
Government Kilpauk Medical College & Hospital
Chennai.

ACKNOWLEDGEMENT

It is my immense pleasure to thank the Dean **Prof.Dr.THIAGAVALLI KIRUBAKARAN, M.D.**, of Kilpauk Medical College and Hospital for kindly permitting me to conduct this study in Medical Department of Government Royapettah Hospital, Chennai.

My heartful gratitude to **Prof.Dr.K.S.SAIKUMAR, M.D.**, Head of the Department of General Medicine for his esteemed guidance and valuable suggestions. It is my privileged duty to thank my teacher-guide **Dr.G.RAJENDRAN, M.D.**, Reader in Medicine, under whom I have the great honour to work as a post graduate student.

I express my sincere and heartful thanks to **(Retd.) Prof.Dr.KALAIVALLI, M.D.**, **Prof.Dr.ARUNACHALAM, D.M., (Cardiology)**, Department of Cardiology, Govt. Royapettah Hospital for their timely suggestions and valuable guidance.

I am greatly indebted to my Unit Asst. Prof. **Dr.T.RAVINDRAN, M.D.**, **Dr.A.NASREEN BEGUM, M.D.**, **Dr.T.RAMESH KUMAR, M.D.**, who have put in countless hours in guiding me in many aspects to conduct this study.

Last but not the least I am thankful to my patients without whom this study could not have been completed.

CONTENTS

INTRODUCTION

Myocardial Infarction generally occurs with the abrupt decrease in coronary blood flow that follows a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis. The injury is produced by or facilitated by factors such as cigarette smoking, hypertension, lipid accumulation, Diabetes and a number of other factors.

A quote from Berkeley heart lab - “Just being an Indian descent puts you at a high risk of coronary artery disease". The pattern of coronary artery disease is indeed changing in India.

It has been reported to be as follows:

a) Coronary artery disease appears a decade earlier in India than other countries.

b) Males are affected more than females

c) Heavy smoking is an important contributing factor

d) Hypertension and Diabetes account for about 40 percent of all cases.

The above findings were based on a clinical study done in Chandigarh in Persons of age greater than 30.

The risk of coronary artery disease in Indians is 3-4 times higher than white Americans, 6 times higher than Chinese and 20 times higher than Japanese Enas EA, Grag A, Davidson NA[12] et al - coronary artery disease risk factors in first generation immigrant Asian Indians to the USA).

Indians are prone as a community to coronary artery disease at a much younger age. The disease pattern is severe and diffuse. Premature coronary artery disease is defined as coronary artery disease occurring before the age of 40 years. Indians are affected by coronary artery disease 5-10 years earlier than the other communities and also they show higher incidence of hospitalization, morbidity, and mortality than other ethnic groups. The above data is based on a study by EnasEA, Dhawan J, Petkar S et al.[3] coronary artery disease in Asian Indians.

In some studies from India, the percentage of patients below the age of 45 years suffering from acute myocardial infarction (AMI) is reported as high as 25-40%[4] Bahuleyan CG Hospital data and Girija G Risk factor profile - Myocardial infarction.

Young patients from other communities do not show extensive disease, whereas in young Indians there is often triple vessel disease with poor prognosis. The post infarction course is also worse in Indians as compared to whites. This is reflected by three time's higher rate of reinfarction and two times higher rate of mortality based on study by Wikinson P Sayer J. Lajik et al & Reddy KS coronary artery disease in India.

The prevalence of coronary artery disease according to the study done by Destefano F, Merrit RK, Anda RF and Bhatia ML[5] shows that it is two times higher (10%) in urban than in rural India. South Indians have higher prevalence 7% in rural and 14% in urban areas. The vulnerability of urban Indians to coronary artery disease is possibly related to different nutritional, environmental and life style factors. The Body Mass Index (BMI) in urban Indians as compared to rural Indians is 24 VS 20 in males and 25 VS 20 in females.

Therefore there has to be a high index of suspicion for coronary artery disease in Indians above the age of thirty. The risk factor evaluation must start earlier. Investigations like tread mill, stress echo, stress thallium and coronary angiography should be more liberally recommended.

This study conducted as a Case-Control retrospective study, aims at evaluating the risk factors involved in the development of myocardial infarction and the clinical spectrum in the younger (≤ 40 yrs) age group.

AIM OF THE STUDY

1. To study the clinical profile of myocardial infarction in young.

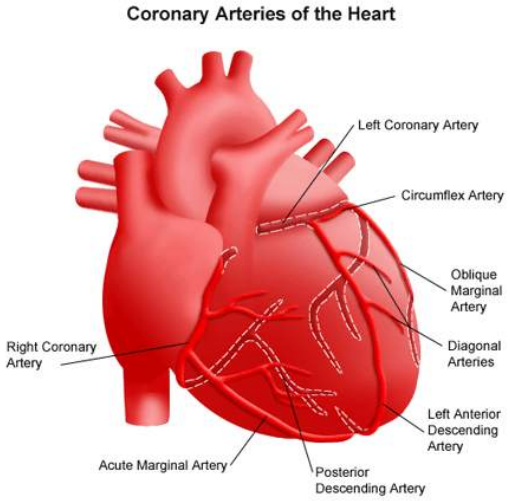
2. To analyse the modifiable and non modifiable risk factors of myocardial infarction in young.

3. To study the multiplicative effects of different combinations of the different Risk factors.

4. To assess the preventative strategy for myocardial infarction.

REVIEW OF LITERATURE

Anatomy of Coronary Circulation



The two coronary arteries that supply the myocardium arise from the sinuses which are situated behind two cusps of the aortic valve at the root of the aorta[6]. The right coronary artery has a greater flow in 50% of individuals and the left has a greater flow in 20% and the flow is equal in 30%.

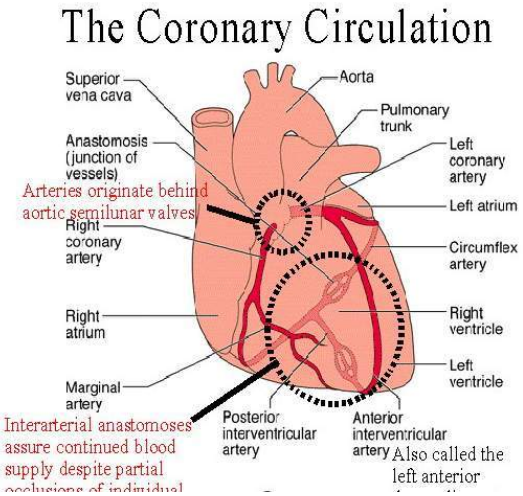
The left anterior descending Coronary Artery (40 to 50%)	-	Anterior wall of the left ventricle and anterior 2/3 of the interventricular septum.
Left circumflex coronary Artery (15 to 20%)	-	Lateral wall of the left ventricle.
Right Coronary Artery (30% to 40%)	-	Inferoposterior wall of the left ventricle, Posterior 1/3 of the Interventricular septum, Right atrium & right ventricle

PRESSURE GRADIENT AND FLOW IN CORONARY VESSELS

The heart is a muscle that, like skeletal muscle compresses its blood vessels when it contracts. The pressure inside the ventricle is slightly higher than in the aorta in systole, hence flow occurs in the arteries supplying the subendocardial portion of the left ventricle only during diastole[7]. Because there is no blood supply during systole in the subendocardial portion of the left ventricle, this region is prone to ischemic damage and is the most common site of Myocardial Infarction. On the other hand as the right ventricle pressure difference with aorta is greater, the flow in right coronary artery is not reduced in systole[2].

CORONARY ANASTOMOSIS

Anastomosis between branches of coronary arteries, subepicardial or myocardial and between the extra cardiac vessels are of prime importance. Clinical experience suggests that anastomosis cannot rapidly provide collateral routes sufficient to circumvent sudden coronary obstruction. It is hence traditional to regard coronary arteries as end arteries.



MYOCARDIAL INFARCTION DUE TO CORONARY ATHEROSCLEROSIS

Myocardial infarction generally occurs with the abrupt decrease in Coronary blood flow that follows a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis.

CORONARY ATHEROSCLEROSIS

The earliest lesions of atherosclerosis can be found in young children and infants in the form of fatty streak whereas the advanced lesion, the fibrous plaques, generally appears during early adulthood and progress with age[8].

The abdominal aorta is involved earliest. The aorta is usually most heavily involved at or near the orifice of its branches (particularly at the level of coronary arteries)[8].

In coronary arteries, raised lesions are most prominent in the main stems, the highest incidence being a short distance beyond the ostia. Atherosclerosis is usually always found in the epicardial (extramural) portions of the vessels, while the intramural coronary arteries are spared, coronary atherosclerosis is often diffuse. The degree to which the lumen is narrowed varies, but once the process has commenced, all the intima of the extramural portions of the vessels are usually involved. Typical atheromatous fibrous plaques also develop in saphenous vein, which is used for aortocoronary by pass graft.

HYPOTHESES OF ATHEROSCLEROSIS

The response to Injury Hypothesis

The endothelial lining cells are exposed to repeated or continuous insults. Dysfunctional endothelial cells at the susceptible sites in the arterial tree would lead to exposure of the subendothelial tissue to increased concentrations of plasma constituents. This triggers a sequence of events including monocytes and platelet adherence, migration, platelet aggregation and formation of microthrombi and release of secretory products. This causes proliferation of smooth muscle cells at these sites of injury. Monocytes become transformed to foam cells. Thus a well developed plaque is formed[9].

Monoclonal Hypothesis

This states that the intimal proliferative lesion results from the multiplication of single, individual smooth muscle cells, as do benign tumors. According to this hypothesis, the intimal smooth muscle cell that proliferate to form an atheroma are normally under feedback control by mitosis inhibitors formed by the smooth muscle cells in the contiguous media, and this feedback control system tends to fail with age as these controlling cells die and are not adequately replaced[10].

RISK FACTORS

A number of factors are present more frequently in individuals who develop atherosclerosis much earlier than in general population and these factors have been termed as risk factors.

The risk factor concept implies that a person with at least one risk factor is more likely to develop a clinical atherosclerotic event and is likely to do so earlier than a person with no risk factors. The presence of multiple risk factors further accelerates atherosclerosis.

Hypercholesterolemia, hypertension and cigarette smoking may be the most potent factors for atherosclerosis[11]. Risk factors also vary in terms of their potential reversibility with current techniques of preventive management.

The 27th Bethesda conferences, conducted by the American College of Cardiology placed the risk factors into four categories. Modification of which will lower the incidence of coronary artery disease.

These categories are as follows:

CATEGORY I

- Basic research and human observational study indicate a clear causal relationship.
- Intervention data demonstrates the magnitude of the benefit and risk.
- Interventions are cost-effective and have been proved to lower coronary artery disease risk.

    1. Cigarette Smoking
    2. LDL Cholesterol
    3. High fat / high cholesterol diet
    4. Hypertension
    5. Left Ventricular Hypertrophy

CATEGORY II

- Studies indicate a causal relationship.
- Intervention data for large scale trials are limited.
- Cost effectiveness not ascertained but likely to lower coronary artery disease

1. Diabetes Mellitus
2. Physical inactivity
3. HDL Cholesterol
4. Triglycerides, LDL cholesterol
5. Obesity
6. Post menopausal status

CATEGORY III

- Studies indicate association
- Interventions have not been tested adequately
- Might lower risk

1. Psychosocial factors
2. Lipoprotein (a)
3. Homocysteine
4. Oxidative stress
5. No alcohol consumption

CATEGORY IV

- Factors associated with coronary artery disease risk, but cannot be modified.

    1. Age
    2. Male gender
    3. Low socio economic status
    4. Family history of early onset of coronary artery disease

SMOKING

Cigarette smoking is the single most important modifiable risk factor for coronary artery disease.

The surgeon general's 1989 report presented definitive data from observational case - control and cohort studies done largely among men that demonstrated that smoking increases the coronary artery disease mortality by 50% and it also doubles the incidence of coronary artery disease. The risk increases with advancing age, duration of smoking and number of cigarettes smoked.

Even among non-smokers, passive exposure increases the incidence of coronary artery disease. Studies suggest that compared with non smokers those who consume 20 or more cigarettes daily have a twofold to threefold increase in total coronary heart disease[12]. Glaniz SA, Parmley WW et al.

Smoking affects atherothrombosis by several mechanisms. In addition to accelerating atherosclerotic progression, long term smoking may enhance oxidation of LDL - C (Low Density Lipoprotein) and reduce levels of HDL - C (High Density Lipoprotein). Smoking also impairs endothelium - dependent coronary artery vasodilatation, increases inflammatory markers such as CRP (C Reactive Protein), soluble intercellular adhesion molecule (ICAM-1) and fibrinogen which causes spontaneous platelet aggregation and increases monocyte adhesion to endothelial cells. Compared with non - smokers, Smokers have an increased prevalence of coronary spasm and may have reduced thresholds for ventricular arrhythmia.

In the first world countries, smoking has significantly decreased and is socially looked down upon. In contrast, in India smoking is increasing particularly in the younger generation. As the demand is falling in the west, tobacco traders are dumping this atherogenic agent in the Indian market. In the seventies tobacco consumption was 0.7 Kg/year/adult. Currently in India consumption of tobacco is 6.1% of the world's tobacco, 20% is in the form of cigarettes, 40% as beedies and 40% as smokeless tobacco products. Studies have shown that 40 - 50% of males in India are smokers13. Borgia C, Depada G, Ciasla EK et al.

Cessation of cigarette consumption constitutes the single most important intervention in preventive cardiology. Smoking cessation alone reduces the risk of a first heart attack by nearly 65 percent.

HYPERLIPIDEMIA

Both hypercholesterolemia and hypertriglyceridemia are important factors for coronary artery disease. A 10 percent increase in serum cholesterol is results in 20 to 30 percent increase in the risk of coronary artery disease. If it occurs early in life, may be associated with higher increase in risk[14] Antanio M, Gotto JR et al.

The increase in cholesterol in associated mainly with a raise in LDL concentrations; the increase in triglycerides are associated with a raise in VLDL and remnants of their catabolism (mainly IDL). Metabolic mechanisms have been postulated whereby abdominal obesity, which is associated with insulin resistance and compensatory hyperinsulinemia, promotes enhanced production of triglycerides and cholesterol rich lipoproteins by the liver and consequent hyperlipidemia, hypertension and hyperglycemia, the so called insulin resistance syndrome[15]. John A Farner, Anntanio M, Gotto JR et al.

This is associated unequivocally with increased incidence of premature ischemic heart disease. In Framingham Study cholesterol level in men below the age of 40 was closely related to the future development of ischemic heart disease. This relation was less pronounced in older age group. In multiple Risk Factor Intervention Trial men with cholesterol above 240 mg/dl has more than a threefold increases in the incidence of fatal ischemic heart disease compared to men with cholesterol below 200 mg/dl. This may be associated with premature atherosclerosis in people with diabetics, hypertensive and with chronic smoking.

According to studies by Moorjani S et al[16], in Indian patients with coronary artery disease high triglycerides level is found more often than high cholesterol levels. Triglycerides bring change in LDL particle size, density, distribution and composition producing smaller, denser, and more atherogenic particles.

Jeppesen J, Hein H, Suadiciani P[16] state that an increase in triglycerides by 90mg/dl has the same effect on coronary atherosclerosis as an increase in age by 10 years.

Earlier there has been an under emphasis on the significance of triglycerides as risk factor for coronary artery disease. Indians worldwide have demonstrated a triad of high triglycerides with high LDL-C levels and low HDL levels.

Studies by Kishore S et al state that higher level of apolipoprotein B is reported in 1/3 of Indian males. This factor in combination with low levels of HDL and hypertriglyceridemia results in the formation of small dense LDL. This increases the risk of coronary artery disease more than three fold.

The LDL - cholesterol types are described as phenotypes A, B or C, which are genetically determined. Patients with LDL - phenotype B have predominantly small and dense LDL particle which constitute an important risk factor for coronary artery disease. A 75% prevalence of phenotype B is seen in Asian Indians in contrast to 25% in white population (Enas EA, Salim Yusuf).

Clear benefits have been demonstrated for dietary and pharmacological treatments that lower serum cholesterol. Treatment aimed at lowering serum cholesterol by 10 percent reduces the risk of coronary artery disease by 15 percent.

HYPERTENSION

High blood pressure is an important risk factor for atherosclerosis leading to ischemic heart disease. The risk increases progressively with increasing Blood pressure.

In a study by Dwivedi[17] et al Hypertension was detected in 51.42% of Indian Myocardial Infarction patients. Faulty life style, particularly genetic predisposition plays an important role in young hypertension as well as in premature atherosclerosis.

In the Framingham study, ischemic heart disease incidence in middle aged men with blood pressure exceeding 160/95 was more than five times than in normotensives. Hypertensive men and women were equally affected particularly when their diastolic pressure was high.

An early Meta analysis that evaluated over 6500 cardiovascular events found a 27 percent increase in the incidence of coronary heart disease and a 42 percent increase in risk of ischemic stroke for every 7 mm Hg elevation of diastolic blood pressure.

Even among individuals without diastolic hypertension, isolated increase in the systolic pressure is a risk factor. It markedly increases the risk for both Non-Fatal Myocardial Infarction (NFMI) and cardiovascular death.

Pharmacological reductions in diastolic BP of about 5 - 6 mm Hg appear to reduce the risk of stroke by over 40 percent, the risk of vascular mortality by 21 percent and the risk of coronary heart disease by 14 percent (Mac Mohan et al).

In India the incidence of hypertension is increasing alarmingly with rapid urbanization. Prevalence of hypertension is increasing in urban population, as compared to rural population in India. In metropolitan cities the prevalence is as high as 11% - 27%.

The age - associated blood pressure increase might be related to physical activity or dietary factors, particularly sodium and total caloric content.

Number of studies done all over the world head conclusively proved that programmes of blood pressure control had consistently been found cost effective.

DIABETES AND INSULIN RESISTANCE

Studies in a variety of populations have shown an association of hyperglycemia with clinically evident atherosclerotic disease, suggesting a role of hyperglycemia in atherogenesis. In both insulin dependent and non insulin dependent diabetics, there is at least a twofold increase in the incidence of myocardial infarction compares to non diabetics. The risk is markedly increased in younger diabetics. Recent data indicate that about one third of patients with IDDM (Insulin Dependent Diabetes Mellitus) die of ischemic heart disease by the age 55. Diabetic women are even more prone to ischemic heart disease than men. Diabetics have a three to five fold increase in ischemic heart disease mortality.

The risk of coronary disease among premenopausal diabetic women resembles that of non-diabetic men, indicating that diabetes largely mitigates the protective effects of female gender.

Recent studies corroborate the role of insulin resistance as an independent risk factor for atherothrombosis. This finding has promoted recommendations for increased surveillance for the Insulin Resistance Syndrome[18,19] a cluster of glucose intolerance and hyperinsulinemia accompanied by hypertriglyceridemia, low HDL level and a predominance of small dense LDL particles. Insulin resistance also produces a prothrombotic state due to increased level of PAI - I and fibrinogen

Diabetic patients have markedly impaired endothelial and smooth muscles function and appear to have increased leukocytes adhesion to vascular endothelium, a critical early step in atherogenesis.

Age adjusted rates for coronary artery disease are two to three times higher in diabetic men and in diabetic women the incidence is seven times higher when compared to non diabetics.

In the Danish Sterno Hospital study mortality from myocardial infarction alone was 12.5% after 35 years of diabetes regardless of the age of onset. Thus individuals with diabetes must be considered at high risk for coronary artery disease regardless of the presence or absence of other risk factors.

PHYSICAL INACTIVITY

Physical activity is an exceptionally common modifiable risk factor for coronary artery disease. In a study of all coronary artery disease deaths in USA in 1986 Physical inactivity accounted for more than one third of cases.

Morris and Colleagues first reported in the mid - 1950 that coronary artery disease rates were lower in people who were involved in various physical activities than those with sedentary life style.

The exact mechanism by which the physical activity operates to decrease the death from ischemic heart disease or possibly to decrease atherogenesis is not known. The possible mechanism is that decreases hyperlipidemia by increasing caloric expenditure. Physical activity also increases HDL. The physical activity acts primarily by its beneficial effect on body weight, blood pressure, serum cholesterol and glucose tolerance.

The risk of coronary artery disease in sedentary individuals was almost twice that of active individuals after controlling other coronary risk factors[20]. Long-term prospective studies of men and women consistently demonstrate that regular physical activity protects against death from coronary artery disease. These benefits apply to activities as simple as brisk walking which has been shown to reduce the risk of coronary artery disease in women and men as well as the risk of type of type II diabetes mellitus[21].

OBESITY

Body fat and its distribution are significantly related to the development of coronary artery disease and other chronic diseases. Coronary artery disease normality rates rise in a linear fashion with obesity. Men with a BMI (body mass Index) of 26 Kg/m^2 or more have a 2.5 fold increase in fatal coronary artery disease compared with those with a BMI of less than 22.5 kg/m^2.

Women aged 30 to 55 with a BMI of 29 kg/m^2 or more have four fold increases in fatal coronary artery event Mansan et al[22].

Obesity produces an increase in cardiac mass and blood volume. Stroke volume and cardiac output are elevated almost in proportion to excess weight. Obesity is also associated with an increase in systolic and diastolic blood pressures. An excess body mass of 10 kg has been shown to increase 5mm Hg in the Systolic blood pressure and 3mm Hg in diastolic pressure (studies by Bethesda National Institutes of Health, 1998).

Obesity is associated with a number of dyslipidemic features. Prospective studies have shown obesity is associated with increased cholesterol levels and a higher incidence of hypertriglyceridemia. An increase in BMI by 10 units is associated with a decrease in HDL cholesterol by 0.28mmol/L in young men and 0.08mmol/L in young women (Anderson et al). Based on cross sectional observations an increase in BMI from 20 kg/m^2 to 30 kg/m^2 is associated with a 0.26 mmol/L increase in LDL cholesterol, which in turn is thought to corresponded to a 10% increase in ischemic heart disease over a five to 10 year period.

An augmented risk of developing diabetes has been noted with obesity. In recent studies 27% of new cases of diabetes were attributed to patients with a weight gain of 5 kg or more. The distribution of body fat may also play a role in the development of coronary artery disease. Abdominal adiposity poses a substantially greater risk in both women and men. Recent studies indicate that the waist to hip ratio, a surrogate for centripetal or abdominal obesity, is an independent marker of vascular risk both in women and older men.

WAIST - HIP RATIO

Waist circumference is the minimum circumference measured between costal margin and iliac crest and hip circumference is measured over the buttocks waist hip circumference ratio more than 0.83 in females and 0.93 in males were taken as abnormal.

WHO has adopted BMI ≥ 25 as overweight Studies by Diwivedi shows that 80.35% males and 91.66% females were centrally obese, 27.5% females who had BMI less than 25 manifested upper segment obesity. Thus waist hip ratio is of great significance.

POSTMENOPAUSAL STATUS

While men exhibit a higher incidence of coronary artery disease at an early age, as well as higher mortality rates from it, the gap narrows substantially after both natural menopause and post bilateral oophorectomy. Women seen with a first time myocardial infarction tend to be older and have higher mortality than in men.

A wide range of factors may explain the increased risk of coronary artery disease after menopause, including adverse changes in lipid and glucose metabolism that result in an increase in LDL cholesterol and a decrease in HDL cholesterol, an increase in glucose intolerance and changes in haemostatic factors and vascular function. Endogenous estrogens appears to play a major role in reducing the risk of coronary artery disease in women, substantial improvement in lipoprotein profile along with a reduction in LDL and an increase in HDL is observed following the initiation of hormone replacement

therapy. There is also a protective effect of vascular function as well as an apparent estrogen related protection of LDL from oxidation. Estrogen also plays a role in maintaining normal hemostasis and improving glucose tolerance.

PSYCHO SOCIAL FACTORS

Psychosocial factors such as depression, absence of social support and anger appear to contribute to an elevated risk of coronary artery disease, although further data are needed to confirm these relationships and establish the efficacy of interventional strategies[23].

When the personality characteristics of coronary artery disease patients were examined, it was found that there was a preponderance of certain type of personality traits, known collectively as coronary prone type A behaviour (Friedman & Roseman).

The type A behaviour is characterized by

1. Time Urgency.

2. Excessive Competitiveness and Hostility.

Overall, there is a chronic struggle to achieve or complete a large number of tasks, working against the limits of time available.

In contrast type B personality is just the opposite, characterized by a relaxed unhurried attitude and less vigorous attempts to achieve a goal.

Studies of therapeutic interventions suggest a role for improving psychosocial factors as part of preventive programmes, particularly in secondary prevention. The strongest evidence comes from post myocardial infarction patients.

A recent Meta analysis of 37 small studies of health education and stress management programmes for coronary artery disease patients suggested that such efforts might reduce cardiac mortality by 34% and recurrent myocardial infarction by 29%. This is due to the favorable effects on blood pressure, cholesterol, body weight, smoking behaviour, physical activity and dietary habits.

LIPOPROTEIN A

The normal function of Lp(a) (Lipoprotein a) is unknown, However Lp(a) may inhibit endogenous fibrinolysis by competing with plasminogen for binding on endothelial surface and increase the release of PAI[24].

Lipoprotein-a is now recognized as an independent risk factor for coronary artery disease. It is a genetic risk factor. It is not affected by life style modifications like changes in diet and exercise. Lp-a is ten times more atherogenic than LDL - C.

Lp(a) values are more or less constant in a single individual since birth and hence estimation of Lp(a) once in a life time irrespective of age is enough for cardio vascular risk assessment.

According to studies by Dilip K.Mukherjee et al.[25] Lp(a) values were higher amongst both Indian boys and girls and more so amongst girls compared to boys.

Lipoprotein (a) Level in	Boys	19.38 ± 5.08 mg %
	Girls	27.26 ± 4.02 mg %

Lp(a) promotes early atherosclerosis and thrombosis. Lp(a) is a stronger risk factor than DM for coronary artery disease in younger women. In Indians, both residents and non residents, the levels of Lp(a) are higher as compared to the whites, suggesting a genetic propensity (Dwington PN et al).

Lp(a) levels according to Sahan et al.[25] of cord blood are higher among Indian newborns than Chinese newborns and this difference is also associated with a four-fold increase in higher coronary artery disease and related mortality in Indians than Chinese in Singapore.

Hunt S et al.[25] in his studies state that Lp(a) levels over 40 mg/dl increases the risk associated with cigarette smoking by 1.9 times, with DM by 3.4 times, with high total cholesterol by 4.2 times, with hypertension by 4.6 times, with high TC/HDL ratio by 6.9 times and with high homocysteinemia by 9.3 times.

Young individuals who have suffered infarction yet who do not have any traditional risk factors are more suitable for assessment of Lipoprotein(a) as this would be the contributing factor.

HOMOCYSTEINE

Homocysteine is a sulfhydryl - containing Amino Acid derived from demethylation of the dietary methionine. Patients with rare inherited defects of methionine metabolism can develop severe hyperhomocystinemia (Plasma levels>100μmol/lt) and can have premature atherothrombosis. The mechanisms that account for these effects remain uncertain but may include endothelial toxicity accelerated oxidation of LDL-C, impairment of endothelial derived relating factor and reduced flow mediated arterial vasodilatation[14] Arneson Refsum H, Bonna HK et al.

In contrast to severe hyper homocystinemia mild to moderate levels of homocysteine (Plasma level > 15 μmol/l) are common in general populations, primarily due to insufficient dietary intake of folic acid. Other patient groups that tend to have elevated homocysteine levels include those with common polymorphisms in the methyltetrahydrofolate reductase gene, those receiving folate antagonists such as methotrexate, and carbamazepine and those with impaired homocysteine metabolism due to hypothyroidism or to renal insufficiency.

A large series of cross sectional and retrospective studies indicate a positive relationship between mild to moderate hyperhomocystinemia and atherosclerosis. On an average, those with plasma levels above 15μmol/l appear to have a relative risk two times higher than individuals with lower levels. However because homocysteine level increases after myocardial infarction and stroke such data cannot be used to establish a cause and effect relationship.

Folic acid given in doses of upto 400µg/day can be expected to reduce homocysteine levels approximately 25 percent, whereas the addition of vitamin B_{12} will likely to reduce the levels by about 7 percent. Because this therapy is inexpensive and has low toxicity in the absence of vitamin B12 deficiency, vitamin supplementation may be a more cost - effective approach for high-risk groups than screening.

OXIDATIVE STRESS

Basic research strongly suggests that oxidative stress plays an important role in the development of atherosclerotic disease and that vit E may delay or prevent various steps in atherosclerosis. However randomized trial data are not yet sufficient to fully assess the role of Vitamin E and Vitamin C or other antioxidants in the primary or secondary prevention of atherosclerotic heart disease.

More such prospective studies will provide valuable information upon which rational clinical decision can be made for both individual and for the general public health care programmes to reduce the incidence of cardiovascular disease. Large consumption of fruits and vegetables which have high contents of micronutrients which act like antioxidants will reduces the risk of chronic diseases.

ALCOHOL CONSUMPTION

Alcohol consumption has complex effects on cardiovascular disease. Studies demonstrate that heavy alcohol intake increases total mortality. While moderate alcohol intake appears to have a protective effect on coronary artery

disease in comparison to non alcoholics Mechanisms underlying the effect of moderate alcohol consumption, defined as one or two drinks daily, including raising HDL levels, as well as improving fibrinolytic capacity and reducing platelet aggregation[34].

With alcohol, the difference between daily intake of small to moderate quantities and large quantities may be the difference between preventing and causing disease for appropriate patients. This can be the basis for the routine preventive counseling in alcoholics. In general, one or two drinks per day may be safe for men. For women, because of their generally smaller BMIs and potential differences in liver metabolism, low levels may be more prudent. However counseling must be individualized.

High alcohol intake is associated with an increased risk of high blood pressure. It appears that alcohol consumption raises systolic pressure more than diastolic. But the finding that blood pressure returns to normal with abstinence suggests that alcohol - induced elevations may not be fixed, and do not necessarily lead to sustained blood pressure elevation.

AGE

Myocardial infarction risk increases with age. In the age group less than 40 years, the highest incidence is in the age of 35 - 40 years. Ischemic heart disease is the major cause of death in males > 35 and all patients > 45.

This can be attributed to the fact that as age increases in both sexes and the blood pressure also increases this may also be due to accumulation of environmental toxins.

FAMILY HISTORY & GENETIC FACTOR

Ischemic heart disease is well recognized as clustering in families. At present, genetic factors contribute 40% and environmental factors contribute 60% in the incidence of ischemic heart disease.

Recent studies have identified a common deletion polymorphism of ACE gene which is associated with increased level of ACE and risk of coronary artery disease. Genetic markers such as LDL receptor, factor V Leiden are also other genetic markers[24]. Genetic factors may play a role by ascertaining direct effects on the arterial cell structure and metabolism as they may act indirectly via such factors as hypertension, increase LDL, diabetes mellitus, obesity, Lipoprotein (a) etc[24].

Coronary artery disease affects Indians 5-10 years earlier than other communities[26]. There is a parallel corollary between coronary artery disease in Indians and the malignant course of rheumatic fever, rheumatic heart disease with associated severe pulmonary hypertension observed by Indian cardiologists[27] Canbian F. Warnet JM, Jaepulson A et al.

SEX

Females are generally more protected from myocardial infarction than men. Women seen with a first myocardial infarction tend to be older than men. The gap narrows substantially after menopause.

In most studies of young myocardial infarction < 40 years, 90% of the cases were male. In females coronary artery disease started 10 years later than men and the incidence raised more towards the menopausal age group.

DIET

Diet plan is an important component of any preventive programmes which leads to weight reduction that can improve dyslipidemia, hypertension and diabetes. One of the most consistent finding in dietary research is that individuals who consume higher amounts of fresh fruits and vegetables have lower rates of heart disease and stroke. Two randomized clinical trials of dietary inventions showed that the risk of cardiac death or acute myocardial infarction was 65% lower. Low fat diet has been shown to reduce the risk of myocardial infarction in healthy individuals and may even cause regression of coronary artery disease. In the opposite direction, saturated and trans-fatty acids appear to increase the coronary artery disease. Antioxidant, vitamins, folate supplement, whole grains, fiber, fish and fish oil seems to have a beneficial effect[28].

The WHO expert committee has recommended the following dietary changes to be appropriate for high risk populations[33].

1. Reduction of fat intake to 20 - 30 percent of total energy intake.

2. Consumption of saturated fats must be limited to less than 10% of total energy intake, some of the reduction in saturated fat may be made up of mono and poly unsaturated fats.

3. A reduction of dietary cholesterol to below 100 mg per 1000 k cal / day.
4. An increase in complex carbohydrate consumption (vegetables, fruits, whole grains and legumes).
5. Reduction of daily salt intake to 5g or less.

Thus a healthy diet can considerably lower the risk of myocardial infarction.

FIBRINOGEN

Plasma fibrinogen critically influences platelet aggregation and blood viscosity, interacts with plasminogen binding and in combination with thrombin mediates the final step in clot formation. In addition, fibrinogen associates positively with age, obesity, smoking, diabetes, and LDL -C and inversely with HDL-C, low alcohol use, physical activity and exercise level[29].

Early reports from Gothenberg, Northwick part and Framingham heart studies all found significant positive associations between fibrinogen and future risk of cardiovascular events. Risk is 1.8 times higher for individuals with high fibrinogen concentration. Plasma viscosity determined in part by fibrinogen level also predicts cardio vascular risk.

Despite major genetic determinants of fibrinogen concentration, substantial variation in plasma levels result from environmental factors. Smoking cessation, increased exercise and weight loss can reduce fibrinogen concentration. Fibric acid derivatives also reduce fibrinogen, apparently through PPAR-α mechanism.

MARKERS OF INFLAMMATION PREDICT FUTURE RISK

These markers include nonspecific acute phase remnants such as hs-CRP, adhesion molecules such as ICAM-1 that are involved in mononuclear cell attachment to the vascular endothelium and cytokines such as interleukin-6 (IL-6) and Tumor Necrosis Factor (TNF). A large body of consistent evidence validates use of acute phase reactants such as CRP and serum amyloid A as markers of risk. Serum amyloid A can bind to HDL particles perhaps rendering them less protective against vascular inflammation.

hs-CRP will likely prove the most clinically useful because it is easy and inexpensive to measure with commercial assay. hs - CRP increases the relative risks of future event of coronary artery disease 3 to 4 times higher than those with lower hs-CRP levels.

INFECTION

Chronic infection with agents such as chlamydia pneumonia, helicobacter pylori, herpes simplex virus or cytomegalovirus virus can lead to systemic inflammation. Such observations have heightened interest in the hypothesis that infection may contribute to coronary risk. Several cross sectional and retrospective studies have identified chlamydia species as well as viral particles in atheromatous lesions[30]. It is important to recognize several mechanisms by which infection might contribute to plaque instability. eg. chlamydia species have been reported to induce macrophage foam cell formation and increases the procoagulant activity of human atheromas which often contain chlamydia, heat shock protein 60, an effector of activation of macrophages, endothelium and matrix metalloproteinase expression.

MINOR RISK FACTORS

Copper deficiency and zinc excess - This leads to secondary hypercholesterolemia leading to increased risk of myocardial infarction.

Water hardness - Water hardness is inversely related to ischemic heart disease. Soft water is generally associated with greater risk of heart disease. Metals like Magnesium, Chromium, Selenium and Zinc have protective effect against myocardial infarction. Cadmium, Manganese and Lead have harmful effects and increases the risk for myocardial infarction.

Heavy coffee drinking-produces tachycardia, arrhythmia and extrasystole and thus predispose individuals to increased risk of myocardial infarction. Lipid rich fractions from boiled coffee increases serum cholesterol level thus increasing the risk for myocardial infarction[28].

Blood Group - Persons with type O blood group appear to be at a lesser risk for myocardial infarction. Research is still ongoing to find out the correlation of such an association.

CLINICAL SPECTRUM OF MYOCARDIAL INFRACTION

Symptoms

Acute Myocardial infarction may commence at any time of the day but have been reported to be preceded by vigorous physical exercise, emotional stress, medical or surgical illness and it occurs mostly in the early mornings. Due to a increase in sympathetic tone and an increased tendency to thrombosis between 6 am to 12 noon.

Pain is the most common presenting symptom, which is deep and visceral pain in central portion of the chest or epigastrium and radiates to the arms. Less common sites of radiation includes abdomen, back, lower jaw and neck. This is accompanied by weakness, sweating, nausea, vomiting, anxiety and sense of impending doom.

Pain may also be absent in some patients. Painless myocardial infarction is common in diabetics and it increases with age. Other less common presentation includes a confessional state and a sensation of profound weakness.

Physical Signs

1. Signs of sympathetic activation - Pallor, sweating, tachycardia.
2. Signs of Vagal activation - Vomiting, bradycardia
3. Signs of Impaired Myocardial function - hypotension, oliguria, cold peripheries, narrow pulse pressure, raised JVP, third heart sound, quiet first heart sound, diffuse apical impulse and lung crepitations.
4. Signs of tissue damage - fever;
5. Signs of complications - eg. Mitral regurgitation, pericarditis (Dressler's Syndrome).

Laboratory Findings

The laboratory tests of value in confirming the diagnosis of myocardial infarction are divided into four groups.

1. Non specific indexes of tissue necrosis and inflammation
2. The electrocardiogram
3. Serum enzyme changes
4. Cardiac imaging

Non Specific Reactions

Polymorphonuclear Leukocytosis appears within hours, lasts for 3 to 7 days, ESR increases - Peaks at first week and remains elevated for 1 to 2 weeks.

Electro Cardiography[31]

The standard 12 lead ECG consists of

Leads, I, II, III aVR, aVL & aVF which are oriented in the frontal plane and are referred to as limb leads.

Leads V_1 - V_6 are oriented in the horizontal plane and are called chest leads.

V_1 - V_2	-	Right ventricle
V_3 - V_4	-	Interventricular septum
V_5 - V_6	-	Anterolateral wall of Left ventricle
L1, aVL	-	High lateral wall
LII, LIII, aVF	-	Inferior wall

Earliest ECG changes are usually ST elevation. Later on there is diminution in the size of R wave and in Transmural (full thickness) infarction - Q wave develops. Subsequently T wave becomes inverted because of change in ventricular repolarization.

In subendocardial infarction there is ST/T wave changes without Q waves or prominent ST elevation often accompanied by some loss of R waves in the leads facing the infarct.

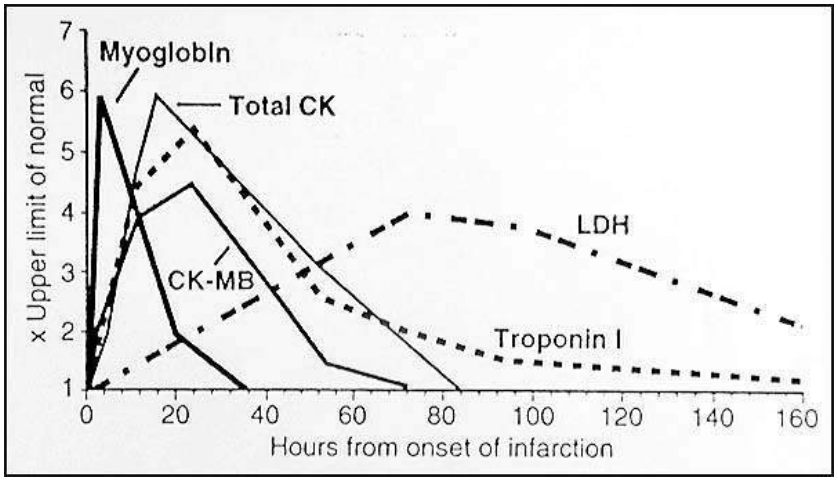
Extensive anterior wall infarction	V_1 - V_6, I, aVL.
Anteroseptal infarction	V_1 - V_4
Anterolateral infarction	V_4 - V_6, aVL, Lead I
Inferior infarction	II, III and aVF
Posterior wall infarction	Reciprocal changes of ST depression and tall R wave in leads V_1 - V_4.
Right Ventricular infarction	V_4 R - ST Segment Elevations

STRESS ELECTROCARDIOGRAPHY

The most widely used test both for the diagnosis of ischemic heart disease and establishing the prognosis involves recording the 12 lead ECG before, during and after exercise on a treadmill or using a bicycle ergometer. Performance is usually symptom limited and the test is discontinued upon evidence of chest discomfort, severe shortness of breath, dizziness, fatigue, ST depression of > 2mm, a fall in systolic BP > 10 mm Hg or the development of a ventricular tachyarrhythmia.

Contraindications to exercise stress testing includes acute myocardial infarction < 4 to 5 days, rest angina < 48 hours, unstable rhythm, severe AS, acute myocarditis, uncontrolled heart failure and active infective endocarditis.

PLASMA ENZYMES[7]



The enzyme most widely used in the detection of myocardial infarction is creatine kinase (CK). More specific and sensitive are troponin T and I. The troponins are released within hours and remain elevated for at least 36 hours. CK starts to rise at 4 - 6 hours, peaks at about 12 hours and falls to normal within 48-72 hours.

AST starts to rise about 12 hours after infarction and reaches a peak on first or second day, returning to normal within 3 to 4 days.

LDH starts to rise after 12 hours reaches a peak after 2 or 3 days and may remain elevated for a week more.

CHEST RADIOGRAPHY

This demonstrates signs of left ventricular failure, cardiomegaly and coexisting cardiac or pericardial diseases.

ECHO

Echo is an invaluable technique for assessing left and right ventricular function. It is useful to detect important complications such as cardiac rupture, VSD, mitral regurgitation and pericardial effusion.

RADIONEUCLOTIDE SCANNING-OTHER IMAGING MODALITIES

Injection of radioisotopes at rest and during stress is performed. In the presence of fixed coronary stenosis there is an inability to increase myocardial perfusion in the territory supplied by the stenotic artery creating a flow difference and inhomogeneous distribution of isotope. Stress - Myocardium perfusion imaging is performed to clarify the significance of an equivocal treadmill exercise electrocardiogram.

CARDIAC CATHETERIZATION AND ANGIOGRAPHY

These remain the only technique that can define coronary anatomy with sufficient precision to support decisions regarding coronary surgery or catheter based interventions in patients with coronary artery disease.

MANAGEMENT[7, 9]

Early management

- Bed rest
- High flow oxygen
- Oral Aspirin, Glycoprotein IIb/IIIa receptor inhibitor
- IV access
- IV analgesic with opiates

- IV antiemetic
- Thrombolysis with IV streptokinase or alteplase
- IV β adrenoreceptor antagonist
- Continuous cardiac monitoring and ECG
- Detect and treat complications early

Oral administration of 160 - 325 mg aspirin daily improves survival (30% reduction in short term mortality).

Coronary thrombolysis helps to restore the coronary patency, preserves the left ventricular function and improves survival. Successful thrombolysis leads to reperfusion with relief of pain, resolution of acute ECG changes and sometimes - transient arrhythmias. The sooner the patient is treated the better the results.

Clinical trials have shown that appropriate use of these drugs can reduce the hospital mortality of myocardial infarction by between 25% and 50% short term mortality was reduced from 13% to 8%.

Streptokinase 1.5 Million units in 100 ml of saline IV over 1 hour is used. It is antigenic and occasionally causes serious allergic manifestation. The drug may cause hypotension and is antigenic. Alteplase is not antigenic and seldom causes hypotension.

ABSOLUTE CONTRAINDICATIONS FOR THROMBOLYSIS

- Active internal bleeding (excluding menses)
- Suspected aortic dissection

- Previous arachnoid or intracerebral hemorrhage
- Major surgery or trauma < 2 weeks
- Recent head injury or known intracranial neoplasm.

RELATIVE CONTRAINDICATIONS FOR THROMBOLYSIS

- Recent Surgery (within 1 month)
- Blood pressure >180/110 mmHg on at least two readings.
- Known bleeding diathesis
- History of CVA
- Active peptic ulcer disease
- Pregnancy
- Hemorrhagic diabetic retinopathy
- Prior exposure to streptokinase

ANGIOPLASTY

Immediate or primary angioplasty of the infarct - related coronary artery is relatively safe and effective alternative to thrombolytic therapy.

ANTICOAGULANTS

Subcutaneous heparin (12500 units twice daily for 7 days) in addition to reinfarction after successful thrombolysis and reduce the risk of thromboembolic complications. Clinical trials have shown that this form of therapy produces a small reduction in short term mortality but also increases the risk of cerebral hemorrhage and of other bleeding complications.

β BLOCKERS, NITRATES AND OTHER AGENTS

Intravenous use of atenolol or metoprolol (β blockers) relieves pain, reduces arrhythmias and improves short term mortality in patients who present within 12 hours of onset of symptoms, but should be avoided if there is heart failure, heart block or severe bradycardia.

Sublingual glyceryl trinitrate is a valuable first aid measure in threatened infarction and IV nitrates are useful for left ventricular failure and the relief of recurrent or persistent ischemic pain. Nitroderm patch which is applied over the skin is also useful in reliving ischemic pain.

ACE INHIBITORS

ACE inhibitors should be prescribed in all hemodynamically stable patients it reduces the ventricular remodeling with subsequent reduction in the risk of the left heart failure. It should be prescribed within 24 hours.

COMPLICATIONS OF MYOCARDIAL INFARCTION

- Ventricular Dysfunction
- Cardiogenic shock
- Mechanical complications
    1. Free wall rupture
    2. VSD
    3. MR

- Arrhythmias

    1. Ventricular premature beat
    2. Ventricular tachycardia/fibrillation
    3. Accelerated idioventricular rhythm
    4. Supraventricular arrhythmias
    5. Sinus bradycardia
    6. Atrioventricular conduction disturbances
    7. Asystole

- Other complications

    1. Recurrent chest discomfort
    2. Pericarditis, pericardial effusion
    3. Thromboembolism
    4. Left ventricular aneurysm
    5. Dressler's Syndrome.

POST INFARCTION RISK STRATIFICATION AND MANAGEMENT

In stable patients sub maximal exercise stress testing should be carried out prior to hospital discharge to detect residual ischemia and ventricular ectopy, and to provide the patient with a guideline for exercise in early recovery period. Alternatively, or in addition a maximal exercise stress test

may be carried out 4 to 6 weeks following infarction. Evaluation of left ventricular function at rest and during exercise is usually warranted as well recognition of a depressed left ventricular ejection fraction by ECHO or radionucleotide ventriculography helps the physician with the selection of pharmacological measures to improve long term outcome. Patients with angina induced at relatively low work loads, those who have a large reversible defect on perfusion imaging or a depressed ejection fraction and demonstrate ischemia, and those in whom exercise provokes symptomatic ventricular arrhythmias should be considered at high risk for recurrent myocardial infarction or death from arrhythmia and cardiac catheterization with coronary angiography or invasive electrophysiological evaluation.

A variety of secondary preventive measures are at least partially responsible for improved long term mortality and morbidity following myocardial infarction. The benefits and use of beta blockers, antiplatelet agents and anticoagulants and ACE inhibitors are discussed above. Finally risk factors for atherosclerosis should be discussed with patient. In particular efforts should be made to ensure the cessation of smoking and the control of hypertension and hyperlipidemia. Additionally regular physical exercise and reduction of emotional stress should be encouraged.

RISK FACTOR ANALYSIS

Studies by Marty AK et al[12] showed the following data about the risk factors for acute myocardial infarction. Smoking was the most common risk factor present.

Smoking	66%
Hyperlipidemia	44%
Stress	40%
Hypertension	28%
Family History	28%
Diabetes Mellitus	18%

Studies by David et al.[33] (1987) in a study on patients of less than 40 years with coronary artery disease showed 95% smokers and 68% had hypercholesterolemia.

Jeyachandran et al.[34] (1987) in a study on premature ischemic heart disease in 150 subjects found that smoking was present in 53% of patients.

Gupta et al.[35] (1987) found in his studies that smoking ranked first among risk factors in young Indian subjects.

Arnold Az's[36] study on coronary artery disease in young women revealed 72% smokers out of which 71% had serum cholesterol levels to be > 200 mg% and 28% were oral contraceptive pill users.

Bergstrand R et al.[37] showed smoking to be the first risk factor for myocardial infarction among men less than 40 years.

Nitter hayge et al.[38] did a study on 66 patients about risk factors the studies revealed, that smoking was present in 86%.

Smoking	86%
Hypercholesterolemia	35%
Systolic hypertension	24%
Diabetes Mellitus	Nil

Studies by Gower Mc et al.[39] in myocardial infarction in those with age <36 years showed that 89% were smokers and 48% had a positive family history of coronary artery disease and 21% had hypertension.

PK Biswas A. Dasbiswas et al.[40] (1994) study on Risk factor and angiographic profile of coronary artery disease in young showed that smokers and hyperlipidemics were the most affected.

PK BISWAS A. DASBISWAS STUDY

Smokers > 10 Cigarettes/day	56.4%
Total cholesterol > 240 mg%	30.6%
HT BP > 160/85 mm Hg	11.3%
Family history of coronary artery disease	11.3%
Obesity (< 20% of ideal body weight)	9.7%
Diabetes Mellitus	6.5%
Hyperuricemia (< 7 mg %)	3.2%

In studies done by[17] Dwivedi, Girish, Chaturvedi, Sanju, Which comprised of 70 young coronary artery disease patients ≤ 40 years there were 56 (80%) males and 14 (20%) females, mostly (67.14%) belonging to 36 - 40 age group. More than half the patients (53%) were from low socioeconomic group.

61.42% were chronic smokers and all of them were males. 18.8% subjects gave history of premature coronary artery disease. Hypertension was detected in 51.42%. Obesity observed in 35.71%. Underweight was noted in 14% of subjects. 80.35% males and 91.66% females manifested upper segment obesity. Hypercholesterolemia was seen in 41.66%. 7.14% were diabetic. Family history of premature coronary artery heart disease in 42.8% and previous hypertension in 42.8% and central obesity (9.166%) were three distinct risk factors in young female subset of patients.

DIWVEDI, GIRISH STUDY[17]

Males	80%
Females	20%
Chronic smoker	61.42%
Premature coronary artery disease	18.8%
Hypertension	51.42%
Obesity	35.71%
Diabetes	7.14%
Family History	42.8%
Hypercholesterolemia	41.66%
Low Socio Economic Status	53%

MATERIALS AND METHODS

The study was conducted at the intensive cardiac care unit and medical wards of Govt. Royapettah Hospital, Kilpauk Medical College during the period of September 2004 to February 2006.

- The study was done as a case control study retrospectively.
- The patients admitted with the clinical features of myocardial infarction with ECG changes suggestive of MI, elevated CKMB and SGOT were taken as cases.
- The patient admitted with the clinical features of Angina and the ECG, CKMB and SGOT were not suggestive of myocardial infarction were taken as controls.
- Two hundred patients were examined and a detailed history with regard to risk factor analysis was made.
- The age groups of less than 40 years were taken for the study.
- The patients were grouped into three categories according to the age 20 to 28, 29 to 34 and 35 to 40.
- The cases and controls were matched according to the age and sex.

OBSERVATIONS

In this study the clinical symptoms like chest pain and its association with sweating, nausea, vomiting, breathlessness and palpitation were all taken into account and the percentage of each was studied.

Physical signs like hypertension, hypotension, raised JVP, edema, S_3, S_4 and crackles, wheeze were all looked for.

Smoking was considered as mild (upto 10 cig/day), moderate (10-15 cigarettes/day) heavy (>15 cigarettes/day)

Hypertension was considered by documented history of hypertension on medication or BP>140/90mm Hg.

Diabetes Mellitus was considered either by documented history of treatment or with a fasting blood sugar level $\geq$ 126 mg % and post prandial blood sugar level $\geq$ 200 mg %.

Serum cholesterol was done for all patients. A lipid profile was also performed.

LDL, HDL & triglyceride levels were taken into study. HDL level was graded as > 50mg/dl, 40-50 mg/dl, <40mg/dl. LDL level should ideally be less than 130 mg/dl, the LDL level was graded as <130,130-160 & > 160 mg/dl.

Questionnaire for personality traits was asked and patient's categorized as type A & type B.

Patients were grouped into three categories according to their age as 20-28, 29-34, and 35-40.

A detailed family history, socioeconomic status, occupation, diet and lifestyle were obtained.

BMI was calculated by using wt/ht^2 and weight/hip ratio was calculated.

RESULT AND OBSERVATION

CLINICAL PRESENTATION - SYMPTOMS

92% of the patients with myocardial infarction had mild to severe chest pain. 65% presented with H/o excessive sweating, 19% had history of nausea & vomiting and 17% associated with breathlessness.

The character of chest pain was assessed to be squeezing, crushing or compressing in nature with radiation in 52% of cases. The radiation was mainly to the left arm, jaw, epigastric region. Nine patients presented with only breathlessness and without chest pain. Six patients had epigastric pain and hiccups, one of the patient presented with TIA with left sided hemiparesis.

Table: 1

CLINICAL PRESENTATION

Symptoms	No. of Cases (Total 100)
Chest pain (angina mild to severe)	92
Chest pain with sweating	65
Radiation +	52
Fatigue	24
Nausea and vomiting	19
Breathlessness	17
Breathlessness without chest pain	9
Palpitation	9
Epigastric pain	6

CLINICAL PRESENTATION - SIGNS

Hypertension was the most common presenting sign. Otherwise 85% of patients had a normal cardiovascular function. JVP was elevated in 7 patients nd S_3 was found to be present in 18 patients. Basal crackles were found in 15 patients, six patients were in hypotension and five patients admitted with associated mitral regurgitation.

Table: 2

Signs	No. of Cases
Normal Cardiovascular functions	85
Hypertension	46
Crackles	15
S_3	18
S_4	6
Hypotension	6
Raised JVP	7
Edema	8
Associated MR	5
Apical shift	2
Wheeze	2

NUMBER OF CASES ACCORDING TO KILLIP'S CLASSIFICATION

Killp gave a classification depend on the status of cardiac pump function, estimated clinically.

Class I - No signs of pulmonary congestion or shock (have excellent prognosis, mortality <5%).

Class II - Moderate heart failure as evident by rales at the lung bases, S_3 gallop, tachypnea or right sided heart failure signs.

Class III - Severe heart failure, pulmonary edema.

Class IV - Shock with systolic pressure < 90mm Hg, evidence of peripheral vasoconstriction, Peripheral cyanosis, mental confusion, oliguria.

Table: 3

NUMBER OF CASES ACCORDING TO KILLIP'S CLASSIFICATION

Class	No. of Cases
I	75
II	18
III	5
IV	2

RISK FACTOR ANALYSIS

AGE

Incidence of myocardial infarction increases with increase in age. In our study 76% of the patients were in the age group of 35-40.

Table: 4

Age	No. of Patients
20-28	9
29-34	15
35-40	76

SEX

Out of the 100 cases under study 89 were males and 11 were females.

Table: 5

Sex	No. of Cases
Males	89
Females	11

AGE: SEX RATIO

In females it was observed that the coronary artery disease started 10 years later than in male and the incidence increases towards menopause.

Table: 6

SEX RATIO IN THE AGE DISTRIBUTION

	Male	Female	Ratio
20-28	9	0	100% male 0% female
29-34	14	1	7% female
35-40	66	10	15% female

There were 89 males and 11 females. Their mean age was 36.66 $\pm$ 4.17 years; females were some what elder to their male counterparts. Most of them belonged to the 35-40 years age group (76%). Youngest male was a 26 years old watchman in private shopping centre, who was a heavy smoker with echo evidence of moderate to severe LV dysfunction. He was a thin individual without evidence of hypertension and diabetes. He had low HDL (29 mg/dl). On a detailed family work up his father (who too was a chronic smoker) was found to have silent coronary artery disease with evidence of dyslipidemia.

FAMILY HISTORY

In our study 72 patients with myocardial infarction had of positive family history and 34 patients without myocardial infarction had a positive family history.

Table: 7

	With MI	**Without MI**
Positive family history	72	34
Negative Family History	28	66

$$\text{Relative Risk} = \frac{\text{Incidence of MI in patients with positive family history}}{\text{Incidence of MI in patients with negative family history}}$$

$$= \frac{a/a+b}{c/c+d} = \frac{72/106}{28/94} = 2.28$$

Thus a positive family history can be associated with a 2 fold increase in risk of developing myocardial infarction.

Family history was found in 72% cases with myocardial infarction. We have included coronary artery disease in all age groups. It is important to remember that positive family history of coronary artery disease in the young has genetic, environmental and life style components which ultimately culminate into young coronary episodes into the family.

SMOKING

Smokers were found to develop myocardial infarction more frequently than non smokers. In our study nearly 80% of smokers developed myocardial infarction.

Table: 8

	With MI	**Without MI**
Smokers	80	36
Non smokers	20	64

$$\text{Relative Risk} = \frac{\text{Incidence of MI in patients who smoke}}{\text{Incidence of MI in patients who do not smoke}}$$

$$= \frac{a/a+b}{c/c+d} = \frac{80/116}{20/84} = 3.06$$

i.e. there is a 3 fold increased risk of developing myocardial infarction among smokers than non smokers.

Out of the 20 non-smokers 11 were females. Considering smoking exclusively in men it was found that 90% men with myocardial infarction <40 were smokers.

Table: 9

Men < 40 with MI	**89**	**%**
Smokers	80	~90%
Non Smokers	9	~10%

HDL level in smokers were calculated. Out of the 80 smokers with myocardial infarction, 68 of them had HDL level lower than 40 mg/dl.

Table: 10

No. of Smokers with HDL > 40 Mg/dl with MI	12	15%
No. of smokers with MI with HDL <40 mg/dl	68	85%

High prevalence of chronic heavy smoking can explain why young subjects who have smoking as a sole conventional risk factor develop premature coronary artery heart disease.

Relative risk of myocardial infarction amongst smokers has been reported to be 3.06. It is also well known that the combination of major risk factors like smoking, hypertension and dyslipidemia in an individual exerts a multiplicative effect on coronary artery disease. The risk increase as much as 9-16 times when all three risk factors are present.

DIABETES MELLITUS

Out of the 100 cases of myocardial infarction, diabetes mellitus was found 14 cases. Even moderate elevation of blood sugar can be associated with increase of coronary artery heart disease.

Table: 11

Total No. of patients with MI	Patients with MI and Diabetes Mellitus	Patient with MI without Diabetes Mellitus
100	14	86

SOCIO ECONOMIC STATUS

53% of patients belonged to low socio economic group, while 40% came from middle income group and only 7% were from upper group. Existence of coronary artery disease in the lower socioeconomic group of people in the metropolis of India is a fact, which is being observed in this country. The scenario has changed considerably since the 70's, when low socioeconomic group of people in rural and semi urban India used to do a lot of physical labour and hard work and incidence of ischemic heart disease was

reported to be low in such cases. These days many of the young able bodied people migrate to neighboring metropolis and adopt faulty life styles which make them prone to coronary artery disease.

80% of young coronary artery disease subjects were chronic beedi smokers. All of them were males and they come from low socioeconomic and middle income segment of the society. Another interesting observation was the fact that only 35% males and smoking as the only conventional risk factors, however many of them had three of four major risk factors.

Table: 12

SES	No. of Patients with MI
Lower Middle	45
Middle	40
Poor	8
Upper Middle	6
Rich	1

LIPID PROFILE

Total cholesterol was measured for all cases. Lipid profile mainly LDL, HDL were measured. Out of the 100 myocardial infarction patients 82% were found to have dyslipidemia. Triglycerides were raised in 45 patients, 72 of them who had a HDL <40mg.

Interestingly 10% of the subjects had HDL $\geq$ 50 mg%

Table: 13

HDL - Cho levels	% of MI patients
≤ 20	2
21-25	14
26-30	11
31-35	13
36-40	32
41-45	14
46-50	4
51-55	6
56-60	2
≥ 61	2

LDL LEVEL

Table: 14

Level of LDL in mg/dl	No. of patients
LDL < 130mg% in MI patients	18
LDL 130-160 mg%	31
LDL > 160mg%	51

It can be seen that the incidence of myocardial infarction increased with increasing levels of LDL Cholesterol.

Patients with LDL cholesterol < 130 mg/dl and ≥ 130 mg/dl were noted in both cases and controls.

Table: 15

	Cases with MI	**Controls without MI**
LDL ≥ 130 mg/dl	82	34
LDL < 130 mg/dl	18	66

$$\text{Relative Risk} = \frac{\text{Incidence of MI in those with LDL} > 130}{\text{Incidence of MI in those with LDL} < 130}$$

$$= \frac{82/116}{18/84} = 3.3$$

(i.e.) patients with LDL ≥130mg% had an approximately 3 fold increase in risk of developing myocardial infarction than patients with LDL<130mg%.

HYPERTENSION

Blood pressure > 140 / 90 was taken as hypertension. 46% of cases with myocardial infarction were found to have a history of Hypertension or were newly diagnosed to have hypertension.

Table: 16

Hypertension	**Myocardial Infarction**	
	Yes	**No**
With HT	46	67
Without HT	54	33

SEDATARY LIFE STYLE

Table: 17

Total No. of Patients with MI	H/O sedentary life style	Active Physical Activity
100	40	60

OBESITY

Obesity was calculated by taking body mass index and waist hip ratio. BMI < 23 consider as desirable. BMI of 23-28.6 was taken as overweight and > 28.6 taken as obese.

Table: 18

BMI	With MI	Without MI
BMI < 23	26	54
BMI 23 to 28.6	38	34
BMI > 28.6	36	12

It is found that 74 patients with myocardial infarction had a BMI above the desirable value clearly indicating that obesity increases the risk of myocardial infarction.

Table: 19

BMI	Myocardial infarction	
	Yes	No
Obesity	36	12
BMI < 28.6	64	88

$$\text{Relative Risk} = \frac{\text{Incidence of MI in those with BMI} > 28.6}{\text{Incidence of MI in those with BMI} < 28.6}$$

$$= \frac{36/48}{64/152} = 1.78$$

Obesity increases the relative risk of myocardial infarction by 1.78 folds.

WAIST - HIP RATIO

Out of 11 women

Table: 20

Waist hip ratio	MI in women	
<0.8	3	27%
>0.8	8	73%

Out of the 89 men

Table: 21

Waist hip ratio	MI in men	
<0.8	29	33%
>0.9	60	67%

It was found that 73% of the women and 67% of men with myocardial infarction had waist hip ratio above the desired level.

- Psychosocial stress was found to be present in 38% of the patients with myocardial infarction. They were all Type A personalities with an evidence of stressful environment.

- Dietary history showed that myocardial infarction was more common in non-vegetarians than vegetarians.

AREAS OF INFARCTION

In our study the most common site of infarction was found to be anterior wall accounting for 55% of cases.

Table: 22

Infarct Site	Cases
Extensive ant wall infarct	55
Anteroseptal	19
Inferolateral	10
Inferior + Right Ventricle	6
Anterior + Interior	6
Inferior + Posterior	2
Anterolateral	2

TYPE OF INFARCTION

More than 62% of patients admitted with myocardial infarction had a Q Wave in ECG.

Table: 23

Q Wave	62%
Non Q Wave	38%

SUMMARISING THE RESULTS

Table: 24

Males	89%
Smoking	80%
Hypercholesterolemia	82%
Family History	72%
Low Socioeconomic status	53%
Hypertension	46%
Sedentary Lifestyle	40%
Psychosocial Stress	38%
Obesity	36%
Diabetes Mellitus	14%

COMBINATION OF RISK FACTORS IN MI

Table: 25

HT + Obesity + DM + Hypercholesterolemia	5
Hypercholesterolemia + Obesity + DM	5
Obesity + Hypertension	26
Obesity + Diabetes mellitus	5
Obesity + Hyper cholesterolemia	30

Some had clustering of several coronary risk factors.

DISCUSSION

In this study of clinical spectrum and risk factors in the young myocardial infarction patients, the commonest symptom was chest pain and the commonest sign was hypertension. Risk factor analysis showed that smoking, hypercholesterolemia, positive family history, hypertension, obesity were frequently associated with young myocardial infarction patients. We are comparing the results of the study with the previous studies.

PRESENTING SYMPTOMS AND SIGNS

In our study most common presenting symptom was chest pain. Other anginal equivalence accounted for 10% of presentation. In our study 14% of the patients were diabetics. Almost all the diabetic patients presented with anginal equivalents. Similar observations have been made in other studies also i.e. Mrgollin Jn, Kinnel WB[41], Fienleich M, et al. (23%) Clinical features of recognized MI silent and symptomatic in AMJ. Cardial 1973; 32:1.

The most common cardiovascular sign was hypertension (46%) similar observations have been made in other studies also i.e. Marty[32] AK Das AK et al (41%), Nitter Hugh[38] et al (24%), Dwivedi[17] et al (51.42%) studies.

RISK FACTORS

Risk Factors in young patients with coronary artery disease: Indian Scenario
Modified from Divivedi, et al., 1997[42, 40, 17]

	VNS 1971 n=28	BOM 1970 n=17	Rhotax 1978 n=65	Old Delhi 1986 n=80	Bikanes 1995 n=45	Chennai 1991 n=217	PK Biswas 1994	Dwivedi 2000 n=70	As per the present study n=100
Overall prevalence of young CAD	12.8	NR	NR	NR	30	NR	NR	NR	NR
Male : Female	8.3:1	All males	NR	9:1	8:1	-	NR	4:1	7.6:1
Family history %	25	12	55	30	29	-	13.3	42.8	70
Smoking %	32	70	70	63	22	10	56.4	61.42	80
Hypertension %	21.4	23.5	15	13	16	24	11.3	51.42	41
Obesity %	35.7	23.5	15	11	NR	NR	9.7	35.71	36
High WHR %	NR	NR	NR	NR	NR	NR	NR	NR	NR
Diabetes Mellitus %	17.8	NR	NR	3	11	18	6.5	7.14	14
High cholesterol %	92.8	70	30	5	3.3	NR	30.6	41.66	79

Smoking:

In our study 80% of the patients were smokers, all of them are males. The probable reason being the atherosclerotic process accelerated by enhanced oxidation of LDL-C and reduced the levels of HDL-C. Smoking all impairs endothelium, increases inflammatory markers and fibrinogen, cause platelet aggregation and increases monocyte adhesion to endothelial cells. In earlier studies to similar observations have been made i.e. Jeyachandran et al.,[34] in 1987 (53%), Gupta[35] et al., 1987, Bergstrand[37] R et al., Gower[39] MC et al., (89%). Dwivedi[17] et al., (61.42%).

Hypercholesterolemia

In our study 82% of patients were found to have dyslipidemia. Hypercholesterolemia accelerates the atherosclerotic process. In addition hypercholesterolemic patients who are associated with obesity, insulin resistance also produces a prothrombotic state due to increased level of PAI - 1 and Fibrinogen. Similar observations have been made in other studies i.e. David[33] JE et al in (68%) 1987, PK Biswas A Dasbiswas S Roy[40] et al., (30.6%) in 1994, VNS[42] study (92.9%) in 1971, BOM[42] study (7-%) in 1970.

Family History

In our study 72% of the patients were found to have positive family history. The genetic factors contribute to the risk of developing ischemic heart disease. The risk is as high as 40%. Common deletion polymorphism of angiotensin converting enzyme (ACE) gene associated with increased level of ACE adds to the risk of coronary artery heart disease. Genetic factor such as LDL receptor, factor V Leiden are also other genetic markers. In earlier studies too similar observation have been made i.e. Marty[32] AK Das AK et al., (28%), PK Biswas A Dasbiswas S Roy et al., (11.3%) in 1994, Dwivedi[17] et al., (42.8%) in 2000, VNS[42] study (25%) in 1971, Rhotak[42] study (55%) in 1978.

Age sex ratio

In our study males were 89% and females were 11% (Male & Female ratio is 8.09:1). Significantly major risk factors like smoking, psychosocial stress and hypertension were high among men as compared to women. In females the risk of developing coronary artery disease started 10 years later

than male. This is presumably due to hormonal factors. The most common age groups affected were between 37 to 40 years. This denotes increasing incidence of myocardial infarction with increase in age. Similar observations have been made in other studies also i.e. Dwivedi et al.[17] (4:1) in 2001, Bikanes[42] study (8:1) in 1994, VNS[42] study (8.3:1) in 1971, Old Delhi[42] study (9:1) in 1986.

Hypertension

In our study 46% of the patients were found to have high blood pressure, the probable reason being the accelerated atherosclerosis, increased left ventricle wall stress, left ventricle tension and stroke work. Other reasons like left ventricular hypertrophy, abnormal coronary flow reserve and abnormal vasomotor response and micro vascular dysfunction also contribute. Similar observation have been found in other studies also i.e. Marty[32] AK Das et al., (28%), Nitter Haugh[38] et al., (24%) Dwivedi[17] et al., (51.42%) in 2000.

Psychosocial Stress

In our study 38% of patients were found to have psychological stress. The probable reason is depression, job stress, social isolation, Type A personality are some of the factors contribute to an elevated risk of Coronary artery heart disease, although further data are needed to confirm this relationship. Similar observations have been made in other study also i.e. Marty[32] AK Das AK et al., (40%).

Obesity

In our study 36% of patients were found to have obesity. Obesity is an important risk factor in the development of coronary heart disease. Obesity is associated with increase in blood volumes, cardiac output and left ventricular filling pressure. When the additional effects of hypertension and glucose tolerance are added, the adverse impact of obesity is even more evident. Obesity especially central obesity is associated with an atherogenic lipid profile. Similar observations have been made in other studies also i.e. Dwivedi[17] et al., (35.71%) in 2000, PK Biswas A (9.7%), VNS[42] study (35.7%), Old Delhi[42] study (11%) in 1986, Rhotak[42] study (15%) in 1978, BOM[42] study (23.5%) in 1970.

Diabetes Mellitus

In our study 14% of patients were found to have diabetes. The reason being it impairs endothelial and smooth muscle function and appears to increase leukocyte adhesion to vascular endothelium, a critical early step in atherogenesis. And also insulin resistance also produces a prothrombotic state due to increased level of PAI- 1 and fibrinogen. Similar observations have been made in other studies also i.e. Marty[32] AK Das AK et al., (18%), PK Biswas A (9.7%), VSN[42] Study (3%) in 1986, Dwivedi[17] et al., (7.14%) in 2000, Chennai[42] Study (18%) in 1991.

CONCLUSION

The following are conclusions that could be inferred from this study on the clinical spectrum and risk factors among young myocardial infarction.

* The most common symptom was chest pain.

* The most common cardiovascular sign was hypertension.

* Most of the patients belonged to Killip's classification-I.

* Most common age group affected was between 36 to 40 years showing that risk of myocardial infarction increase proportionately with increasing age.

* Males were commonly affected especially in the younger age group. Significantly major risk factors like smoking, psychological stress, and hypertension were also evident among men as compared to women. These factors along with hormonal factors contribute the higher proportion of myocardial infarction in young males.

* Females showed an increased risk of myocardial infarction towards the later stages of life presumably due to hormonal factors.

* Risk factors analyses proved that smoking was the single most important risk factor for myocardial infarction.

* Majority of patients with myocardial infarction had dyslipidemia.

* Family history of myocardial infarction is an important risk factor contributing to myocardial infarction in young individuals. This is probably due to an inter play of both genetic and environmental factors.

* Hypertension was closely related with risk of myocardial infarction.

* The obesity was an important risk factor in young myocardial infarction patients probably due to the increasing incidence of sedentary life style.

* The incidence of diabetic mellitus in young myocardial infarction patients was comparatively less than the incidence of other risk factors.

SUMMARY

Chest Pain is the cardinal symptom in young myocardial infarction. Breathlessness, sweating, radiation, vomiting, palpitation, are also common symptoms. Hypertension, S_3, S_4, crackles, were commonly observed signs. On few occasions infarction also occurred in the absence of physical signs or symptoms.

Key factors influencing the development of myocardial infarction were non modifiable risk factors such as age, sex, family history. Smoking is probably the single most important modifiable risk factor. There is a strong relationship between cigarette smoking and coronary artery heart disease in young individuals. The risk is also closely related to plasma LDL cholesterol, and inversely related to HDL cholesterol concentration. Obesity particularly central or truncal is an independent risk factor. Additional risk factors such as diabetes, physical inactivity added to the adverse impact.

The effect of risk factors is multiplicative rather than Additive. Thus people with the combination of risk factors (smoking, hypertension, diabetes) have the greatest risk of developing myocardial infarction.

Prevention can aim at modifying the risk factors like cessation of smoking, reduction of weight, reduction of salt intake, dietary changes, increase physical activity and control of psychosocial stress. This will have a tremendous impact in reducing the incidence of myocardial infarction in the young.

ABBREVIATIONS

Sl.No.	-	Serial Number
Sx	-	Sex

Clinical spectrum - Symptoms and Signs

CP	-	Chest Pain
RA	-	Radiation
S	-	Sweating
N/V	-	Nausea/Vomiting
DWOC	-	Dyspnea without chest pain
DWC	-	Dyspnea with chest pain
F	-	Fatigue
P	-	Palpitation
EP	-	Epigastric pain
H	-	Hypotension
RJ	-	Raised JVP
PE	-	Pedal edema
AS	-	Apical shift
MR	-	Mitral regurgitation
R	-	Rales
W	-	Wheeze

Risk factors

SM	-	Smoking
HT	-	Hypertension
DM	-	Diabetes mellitus
FH	-	Family history
OB	-	Obesity
LDL	-	Low density lipoprotein
HDL	-	High density lipoprotein
LSES	-	Low Socio Economic Status
SL	-	Sedentary Life
PSS	-	Psycho social stress

BIBLIOGRAPHY

1. ENAS - EA Garg - A, Davidson NA et al coronary artery disease on its risk factor sin the first generation immigtsny.

2. ENAS - EA, High rates of coronary artery disease in asian Indians in USA despite intensive modification of life style. What next. Current Science 1998;74-89.

3. ENAS EA, Dhawan J, Petkar S et al. Study on coronary artery disease in Asian Indians.

4. Girija G Risk factors profile of patient with acute myocardial infarction in Vijaya Ragavan G Cardiovascular disease prevention Trivandrum 78-83.

5. Destefano F, Merrit RK, Anda RF Trends in CHD in the USA 1980 through 1989. ARCH Intern med.1993:153"2489-94".

6. Peter Williams, Rogier Warwick, Grays books of anatomy.

7. Eugene braunwald heart diseases.

8. Cotran, Kumar, Robbins book of pathology.

9. Harrison Principles of internal medicine. 15th Edn. 1387-1399.

10. Davidson book of Internal Medicine. 19th Edition.

11. Robert C Sanlant R Wayne Alexander "The Heart".

12. Glantz SA, Parmley WW passive smoking and heart disease, mechanisms and risk, JAMA 273, 279.

13. Borgia C. Depaola G, Ciasla Ek et al cigarette smoking and AMI Minerva cardioangiology. 1992 41 715-722.

14. Antanio M Gotto JR lipid and lipoprotein disorders primer in preventive cardiology Dallas American Heart Association 1994. 281. 1782-7.

15. John A Farner, Anntanio M Gotto JR root factor of coronary artery heart disease 1996. 11:10-14.

16. Moorjani S, et al. Jeppesen J, Hein H, Saradiciani P. et al. of triglycerides level in Indian patients with CAD.

17. Coronary artery disease in the young; Heredo familial or faulty life style or both by : S DWIVEDI, GIRISH DWIVEDI, A CHATURVEDI, SANJIV SHARMA, journal Indian academy of clinical medicine Vol.No.3, Oct - Dec 2000.

18. David J. Maron, Paul M Ridker,Thomas A Pearson Risk factor and prevention of coronary artery disease 1998. 888-910.

19. Weatherall ledingham warwell oxford text book of medicine volume 2.

20. Chaiknounni A choudhanl pomposiello Cbanna A et al., myocardial infarction in Qator the just 2515 patients clinical cardiology 1993 March 16.

21. Praveen Kumar and Michael Clark, "Clinical Medicine" 1994 3rd edition.

22. OB Panlajani sichemic heart diseases API textbook of medicine 5th edition. James T.Willison, Jay N Cohn, CV Medicine. Page No.484-492.

23. Jay H.Stein "Internal Medicine: 1983 first edition.

24. Hamsten A Iselius et al "Genetic and cultural inheritance of serum lipids - low and HDL cholesterol and apolipoproteins AI ALL & B atherosclerosis. BMS 1992: 715-22.

25. Mukherjee et al. Dwington PN, et al. Sahan et al, Hunt et al. Serum lipids, HDL cholesterol and apolipoproteins in atherosclerosis & CAD.

26. Coronary artery disease in young Indian - The Missing Link by HS RISSAM, S KISHORE, N TREHAN Journal, Ialm - Vol5 May 1996.

27. Canbian F Warnet JM Jaepulsan A relation of Parentral history of early myocardial infarction to the level of apoportein B. 1987. 92 112-188.

28. Inger stensvoid et al Cohort study of coffee intake and death from coronary heart disease over 12 years British Medical Journal. 1989;27 38-45.

29. William, "Text book of Endocardiology" 8th edition. 210-222.

30. Janus ED, Postiglione A, Singh RB etal.The modernization of asia "Implication of Coranary heart disease" circulation 1996; 94; 2671-73.

31. Leo Schamroth An introduction to Echocardiography 7th edition.

32. Marty A.K. Das AK et al prognostive significations of risk factors in AMI in young. Indian Heart Journal. 1991 13 108-111.

33. David JE Mallal FJ Cheitlinm ND Coronary artery disease in young patients. Arteriographic and clinical review of 40 cases aged 35 and under American heart journal 1977. 74:318-31.

34. Jeyachandran R Sathayamathy I, Subramanyan K. et al., Coronary artery disease in found Indian Heart Journal. 1996; 37 112-118.

35. Gupta SR, Gupa SK, Reddy KN et al coronary artery disease in young Indian subjects Indian Heart Journal. 1996; 48 241-45.

36. Arnold AZ Moodids Coronary Artery diseases in young women. Risk factor analysis long term follow up cleve clinical journal of medicine. 1999:281 1722-27.

37. Bergstrand R Vedin A wilhelnsson C et al Myocardial infarction among men < 40 british heart jounral. 1989;41-715-22.

38. Nitter Hauge S Erickssen J Thawlow E et al., "Angiographic and risk factor characteristic of subjects with early onset of ischemic heart disease British heart jounal. 1987; 114 4477-82.

39. Gower MC et al Myocardial infarction before age 30 Risk factor and angiographic analysis "American Journal of Cardiology". 1993; 328 538-45.

40. PK Biswar, A Dasbiswas S Roy et al risk factors and angiographic profile of coronary artery disease of young JIMA, 1992 22-26.

41. Mrgolin JN, Kinnel WB[48], Fienleich M, et al. (23%) Clinical features of recognized MI silent and symptomatic in AMJ. Cardial 1973;32:1.

42. Dwivedi S, Anupam P. Chathurvedi A, Cardiovascular risk factor in young coronary artery heart disease patient around East Delhi.South Asian journal preventive Cardiology 1997; 1:21-26.

43. Park's textbook of preventive and social medicine.

44. APICON PG Medicine Class of risk factors in coronary artery disease volume 13. Page No.112.

45. ENAS AE Yousuf et al prevalence of coronary artery disease in asian Indians, Aerican journal of CAHD and malignant coronary artery disease in young Asian Indians. 1992 : 19-56-64.

46. Arneson Refsum H. Bonna HK et al. Serum total homocystine and coronary artery heart disease International Journal of Epidem. 1978 30 2000-02.

47. Cecil textbook of medicine.

48. Jonathan Abraham, Richard C, Partenak Cardiology Clinics Changing the natural history of coronary artery disease Risk factors as their modification. BH3 1995. 74; 318-23.

49. Wikinson P Sayer J. Lajik et al of comparison of case fatality in south asian and white patients after Acute myocardial infarction BMJ 1996; 312 "1330-1333".

50. American heart association, Heart & Stroke Strategical update 1997;26-27.

51. Bahuleyan CG Hospital Data on coronary artery disease North kerala in Vijaya Ragavan G Cardiovascualr disease prevention Trivandrum 54-59.

CLINICAL PROFORMA

1. Name :
2. Age :
3. Sex :
4. Marital Status :
5. Educational Status :
6. Occupation :
7. Income/Year :
8. Residential Address:
9. Height :
10. Weight :
11. BMI (Wt/Ht in M^2) :
12. Waist / Hip Ratio :

CLINICAL PRESENTATION OF ILLNESS

Chest Pain - Characters :

Compressive, Retrosternal, Consistent, Radiating to left arm or root of the neck or back of the chest.

Angina Equivalent :

H/o nausea, vomiting, epigastric pain, jaw pain, neck pain, left arm pain, profuse sweating, fatigue, breathlessness, palpitation, syncope.

Time of onset of symptoms :

Admission Time : Therapeutic window:

Risk Factors Identified

1. Hypertension : How may years:

   On treatment - Yes / No Regular / Irregular Rx

2. Diabetes Mellitus : IDDM / NIDDM (how may years)

   A. Known / B. Newly diagnosed Regular Rx / Irregular Rx

3. Hyperlipidemia No.of years

   Treatment Diet

   Drugs - specify

4. Smoking: Beedies/Cigarettes smoking /Others

   No.of Cigarettes / Day

   No.of years of Smoking

   If stop smoking - How may years back?

5. Alcohol consumption : 1. Quantity
   2. Frequency
   3. Duration

   if stopped - When?

6. Obesity

7. Family H/o ischemic heart disease

8. Sedentary Habit
   (Lack of Exercise)

III. OCCUPATIONAL HISTORY

Presence of Stress in Occupation (Direct)

Stress from Colleagues (either junior staff (or) Senior Staff)

IV. DIET HISTORY

Vegetarian / Non Vegetarian

Frequency Consumption of rice - No.of Times / Day
Duration Type of Cooking Oil used.

Salt Intake

Coffee / Tea

Living Condition : Nuclear Family / Joint Family / Alone
Hut / Tiled House / Independent / Flats
Hygiene / No. of Dependents / Income per month

Menstrual history : Premenopausal/Postmenopausal
Menopause : Years since menopause

Sexual history

Adequacy of Sex/Extramarital relationship

(Presence or absence of stress)

Personality:

Type A	Type B
Perfectionist	Causal Attitude
Very competitive	Not competitive
Always rushes	Never
Impatient when waiting	Can wait
Tries to do many things at a time	Take things one at a time
Express feelings	Does not
Ambitious	Not ambitious

General Examinations:

Conscious: Orientation: Co-operation:

BP PR PR JVP

Anaemia Cyanosis Pedal Edema
Icterus Clubbing

Markers of hypercholesterolemia

Systemic Examination (CVS)

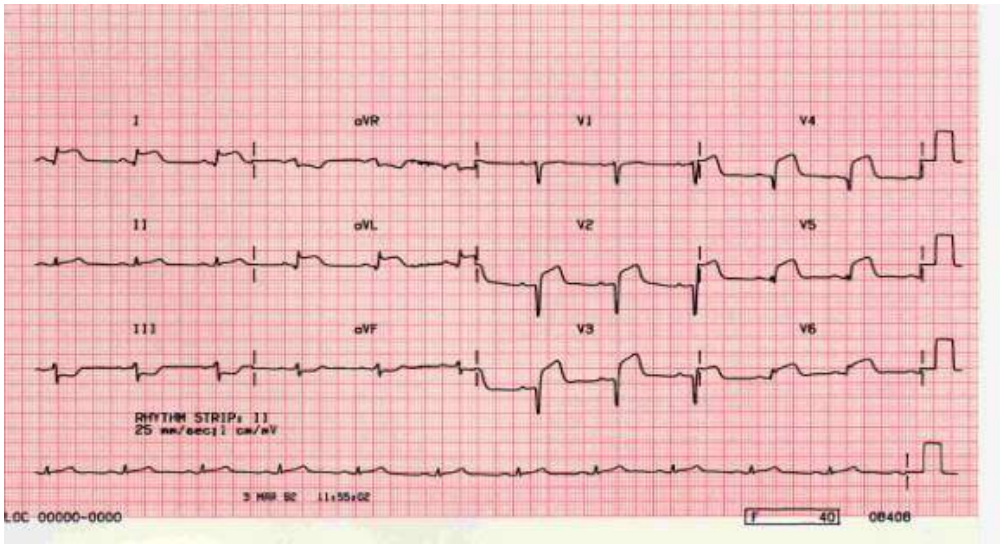
Apical impulse	Site	Abnormal systolic pulsation
	Nature	
Heart Sounds	S_1 & S_2	S_3/S_4
Murmur of MR/VSD		Additional Sounds
Pericardial rub		

Respiratory System (RS)

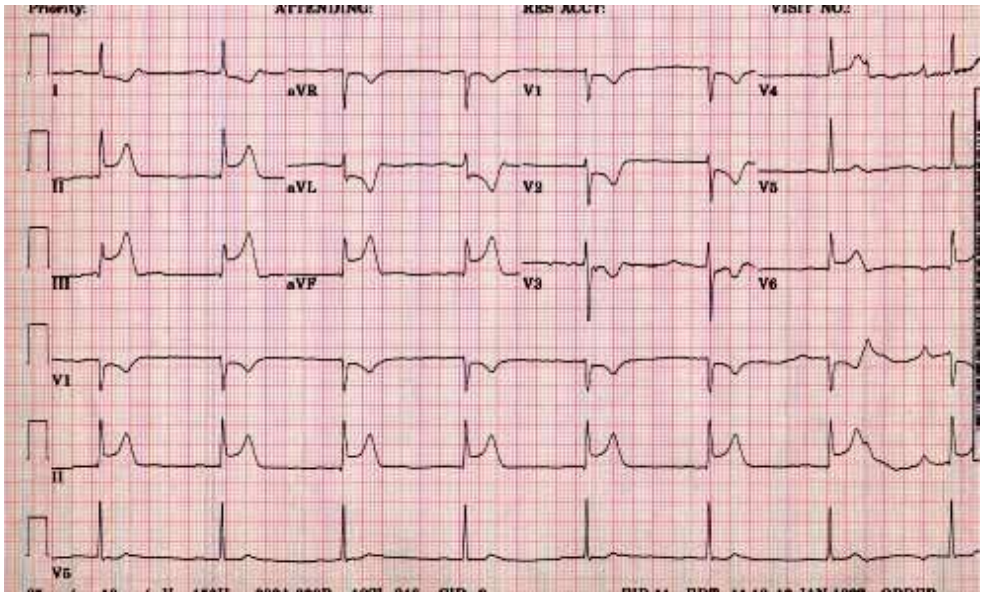
Air entry equal B/L	Adventitious Sounds - Crepitations - Wheeze - Pleural rub

Investigations

1. Urine - Alb
   Sug
   Dep.
2. TC
   DC
   ESR
   Hb%
3. Blood Ureas
   Sugar
   Sr.Creatinine
   Sr.Electrolytes
4. Total Cholesterol — Lipid Profile — Triglycerides, LDL, HDL
5. Cardiac enzymes — SGOT
6. ECG
7. Chest X-ray
8. ECHO

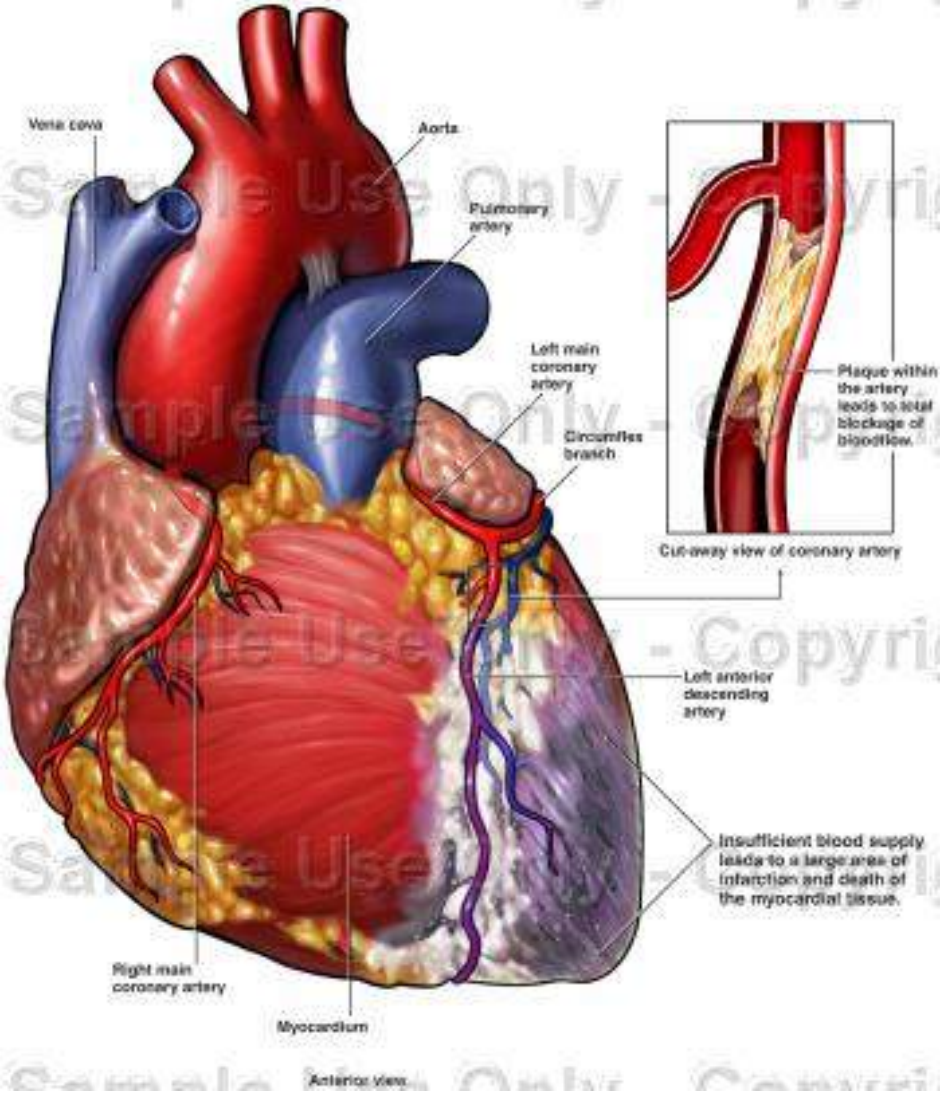


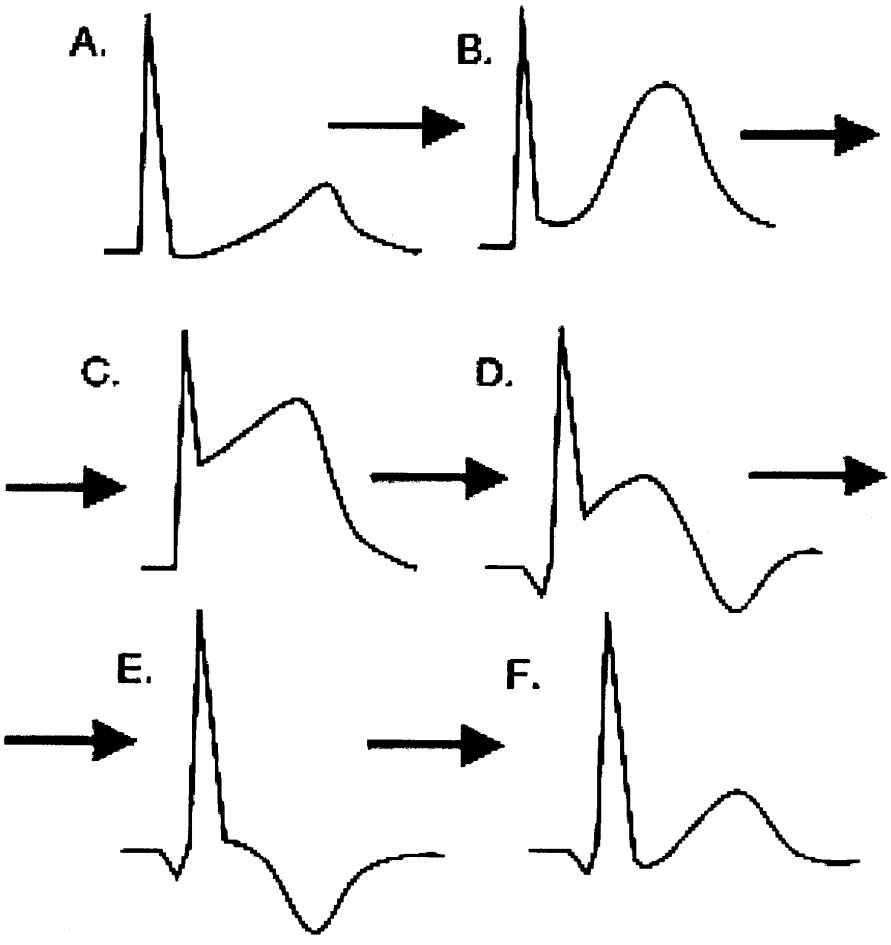
Acute extensive anterior wall infarction



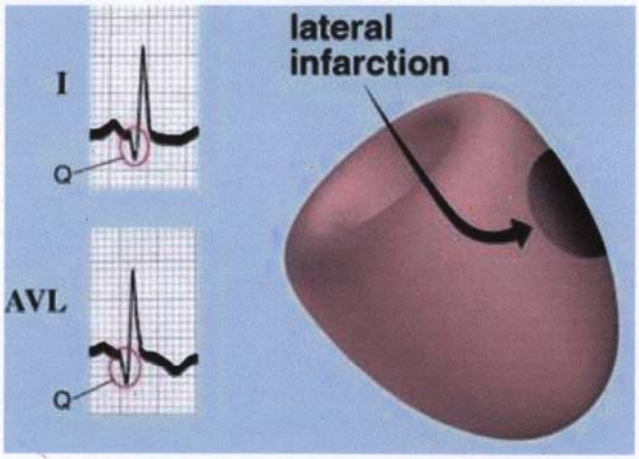
Acute inferior wall infarction

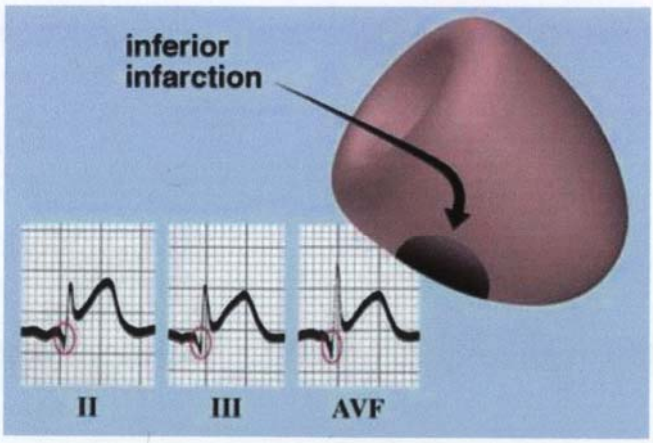
Coronary Artery Stenosis with Subsequent Myocardial Infarction

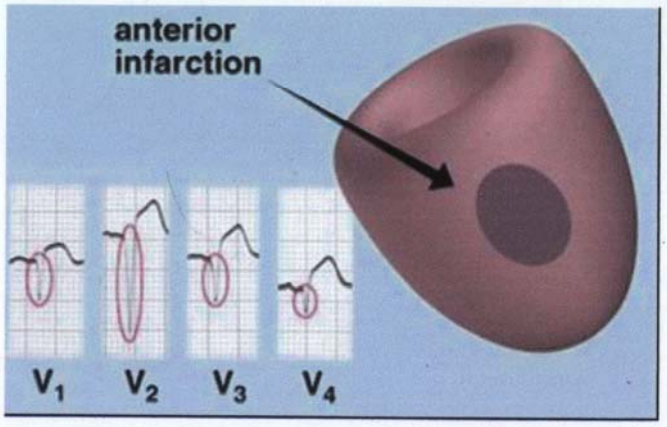


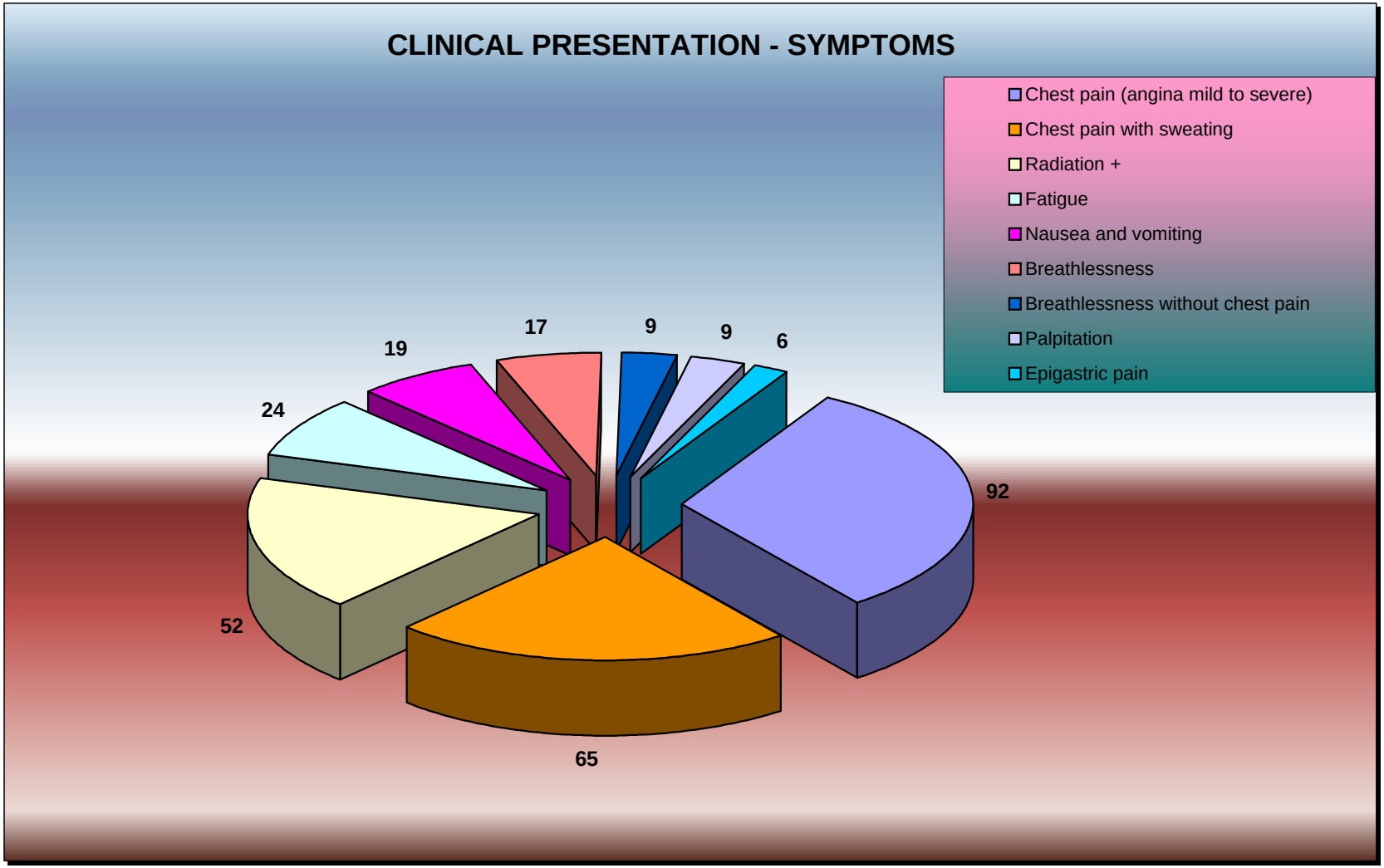


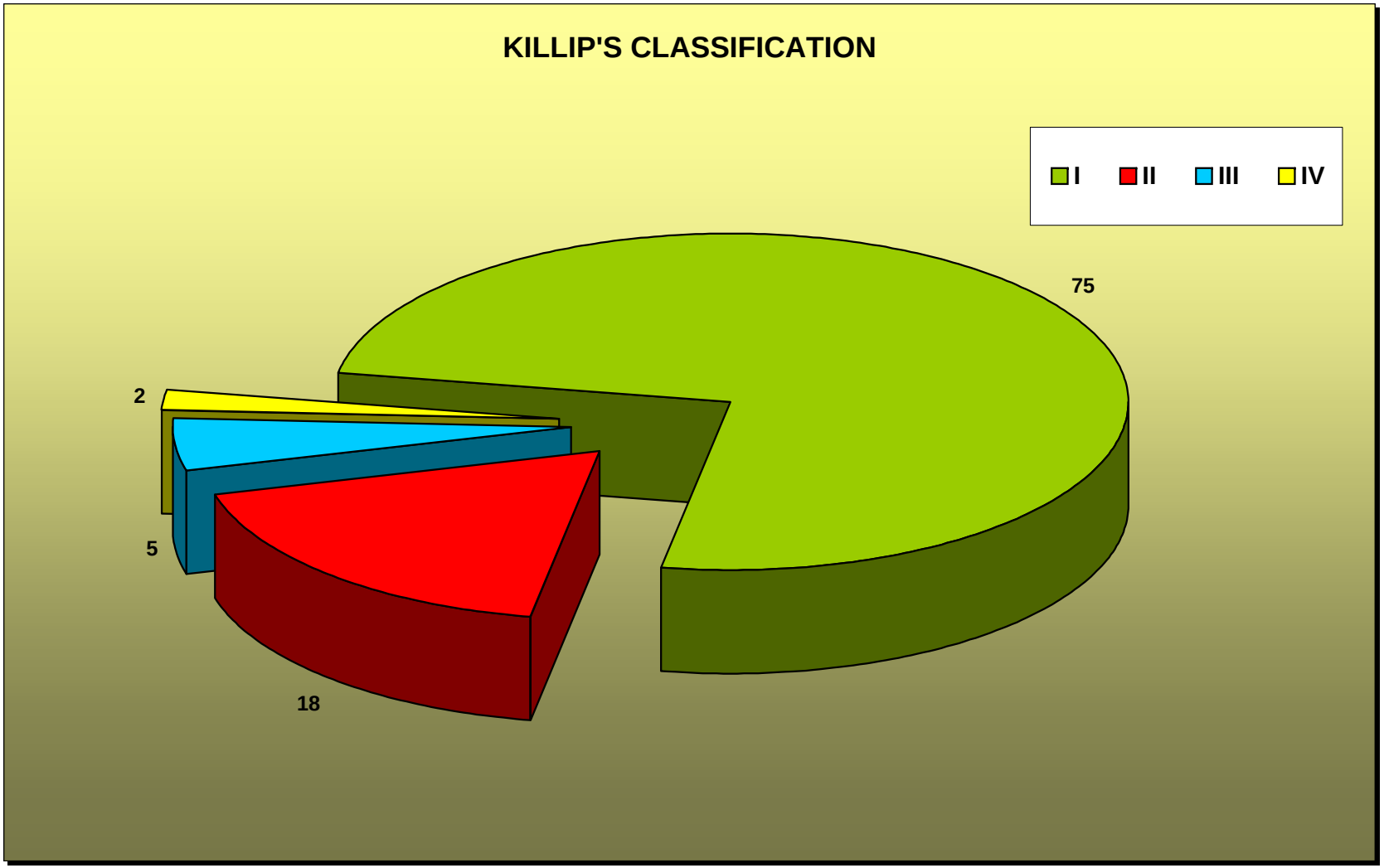
Evolution of Acute MI


lateral
infarction
I
Q
AVL
Q

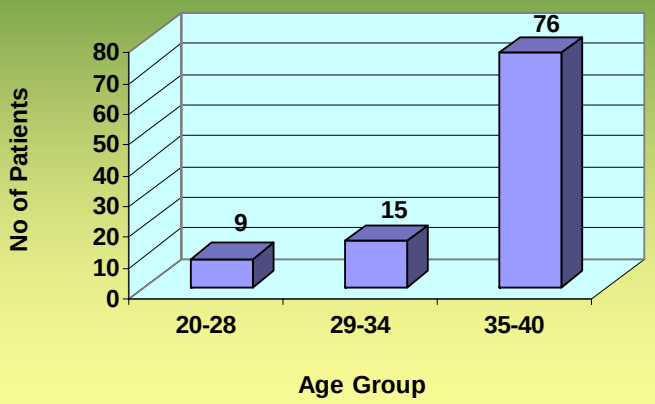

inferior
infarction
II
III
AVF


anterior
infarction
V1
V2
V3
V4

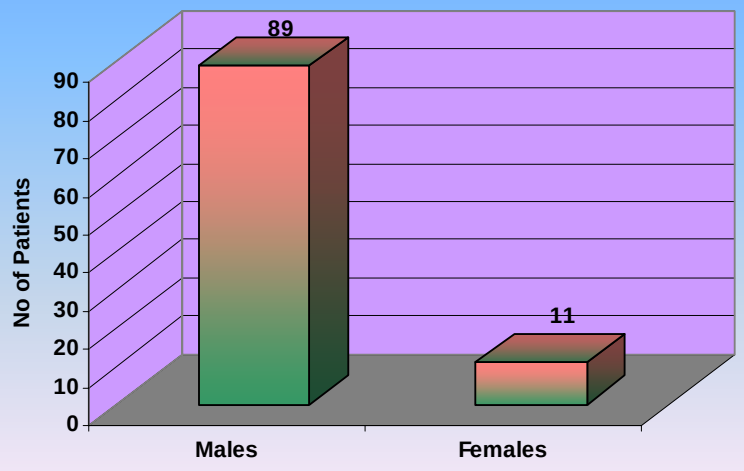

CLINICAL PRESENTATION - SYMPTOMS
Chest pain (angina mild to severe)
Chest pain with sweating
Radiation +
Fatigue
Nausea and vomiting
Breathlessness
Breathlessness without chest pain
Palpitation
Epigastric pain
92
65
52
24
19
17
9
9
6

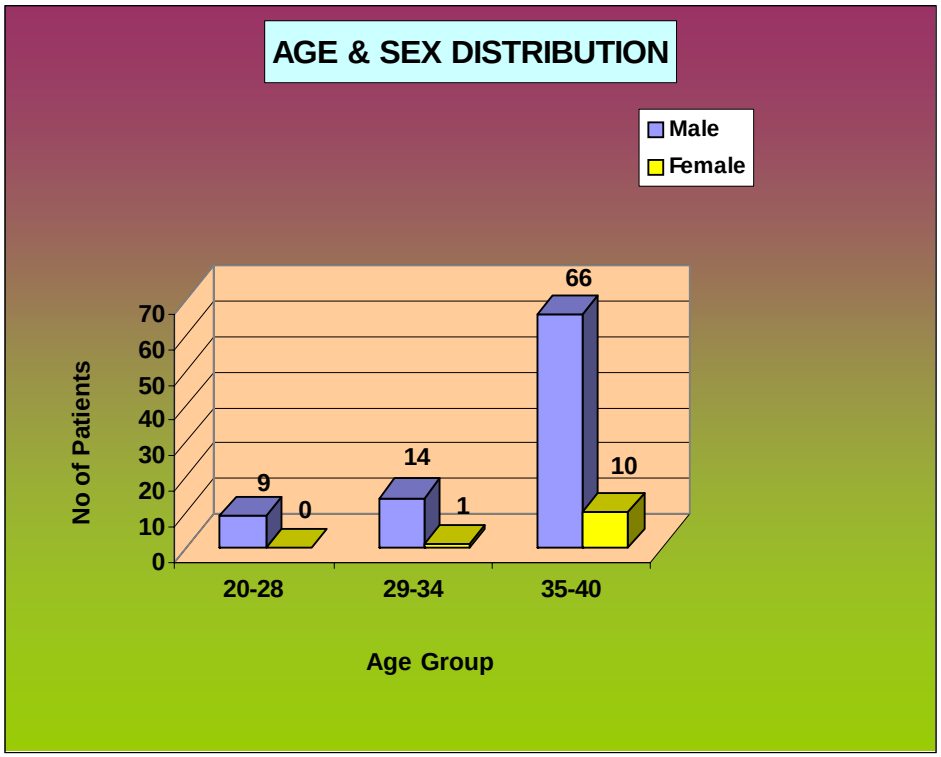

KILLIP'S CLASSIFICATION
I
II
III
IV
75
2
5
18

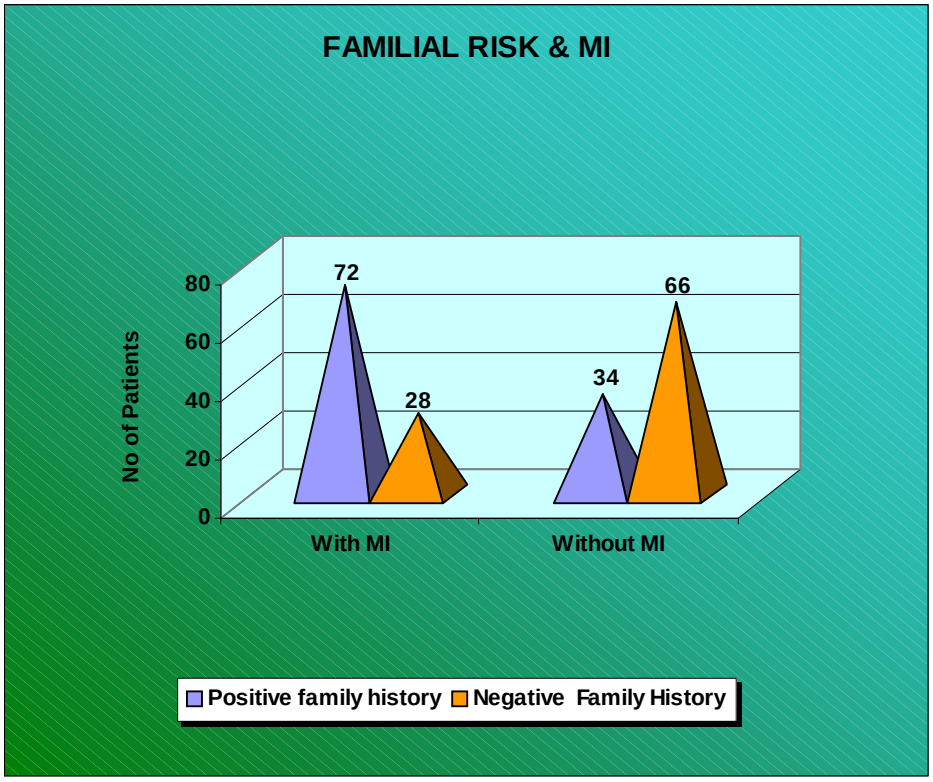
AGE DISRIBUTION

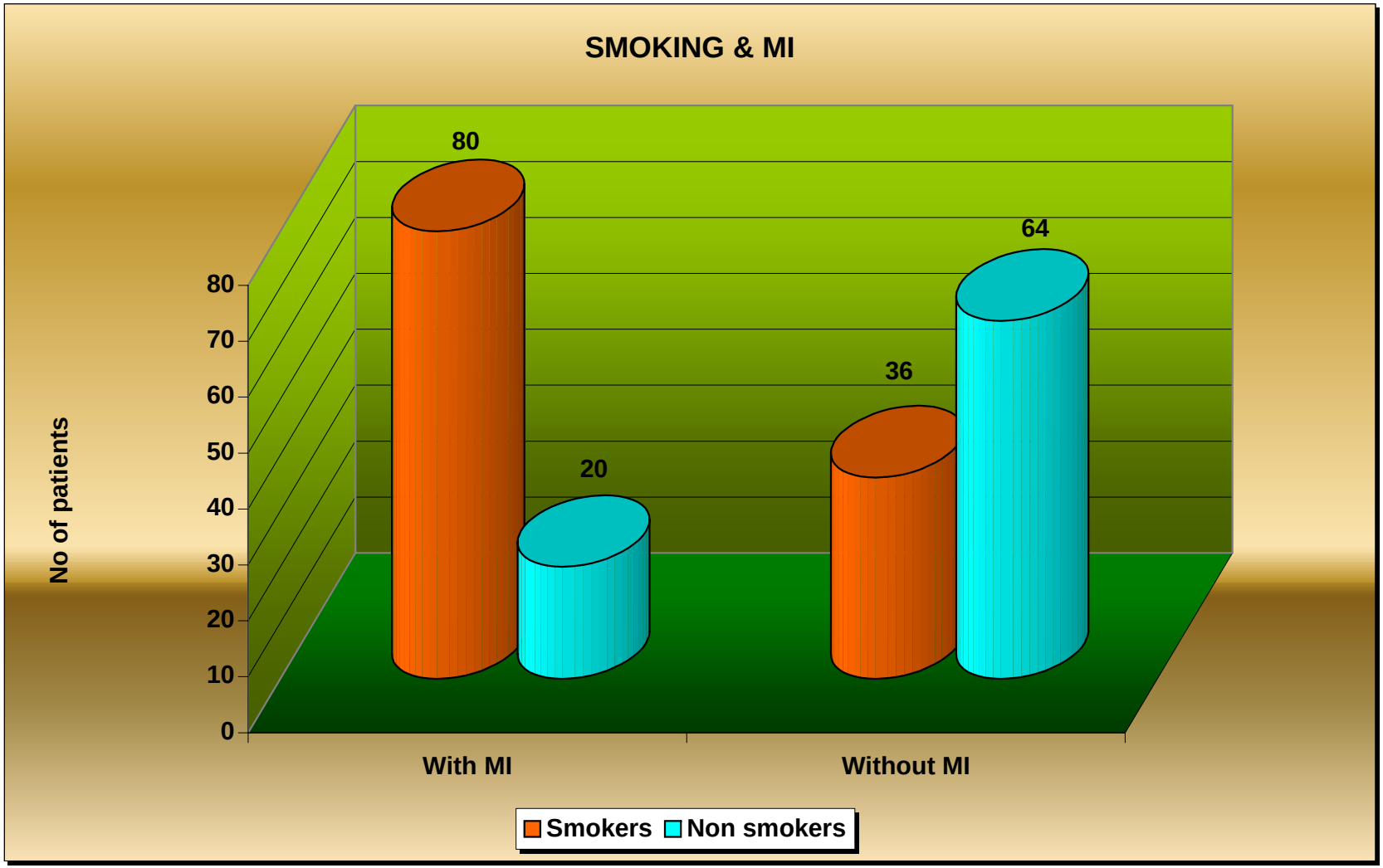


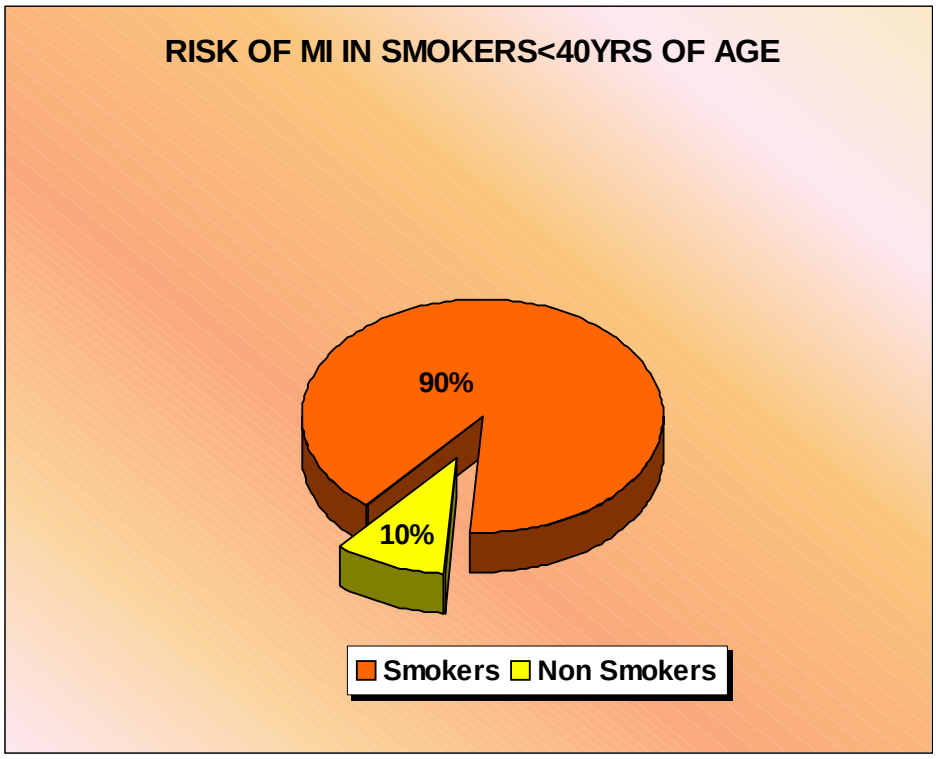
SEX DISTRIBUTION

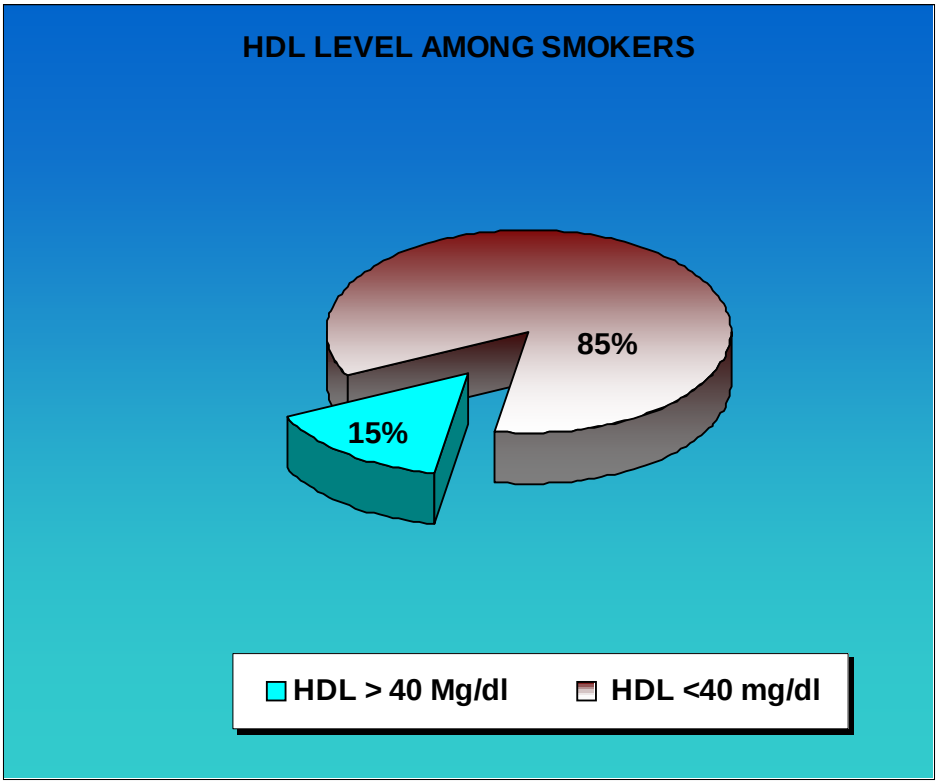


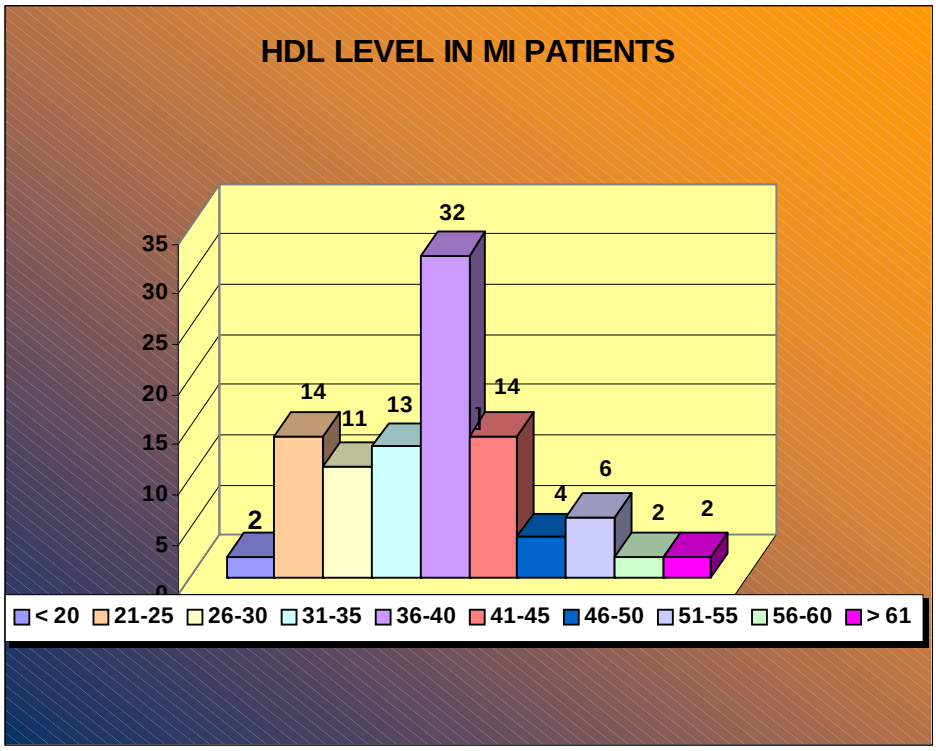

AGE & SEX DISTRIBUTION
Male
Female
No of Patients
70
60
50
40
30
20
10
0
9
0
14
1
66
10
20-28
29-34
35-40
Age Group

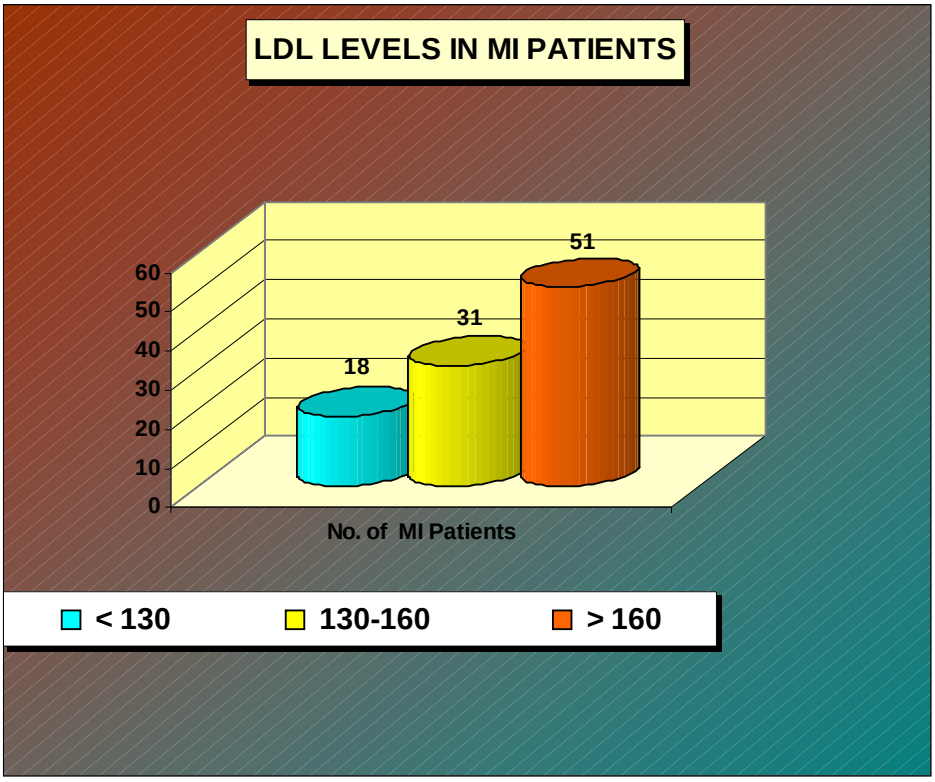

FAMILIAL RISK & MI
No of Patients
80
60
40
20
0
72
28
34
66
With MI
Without MI
Positive family history
Negative Family History


SMOKING & MI
No of patients
80
70
60
50
40
30
20
10
0
80
20
36
64
With MI
Without MI
Smokers
Non smokers

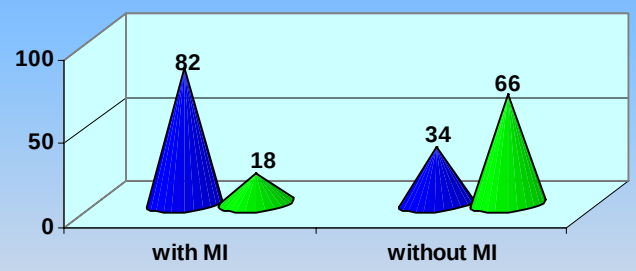

RISK OF MI IN SMOKERS<40YRS OF AGE
90%
10%
Smokers
Non Smokers


HDL LEVEL AMONG SMOKERS
85%
15%
HDL > 40 Mg/dl
HDL <40 mg/dl


HDL LEVEL IN MI PATIENTS
35
30
25
20
15
10
5
0
2
14
11
13
32
14
4
6
2
2
< 20
21-25
26-30
31-35
36-40
41-45
46-50
51-55
56-60
> 61

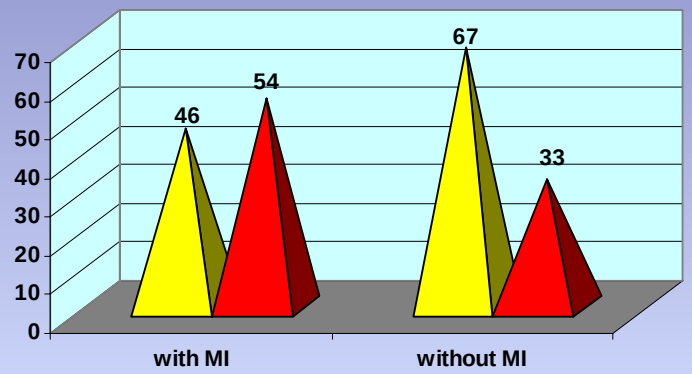

LDL LEVELS IN MI PATIENTS
60
50
40
30
20
10
0
18
31
51
No. of MI Patients
< 130
130-160
> 160

LDL LEVELS IN PATIENTS WITH & WITHOUT MI

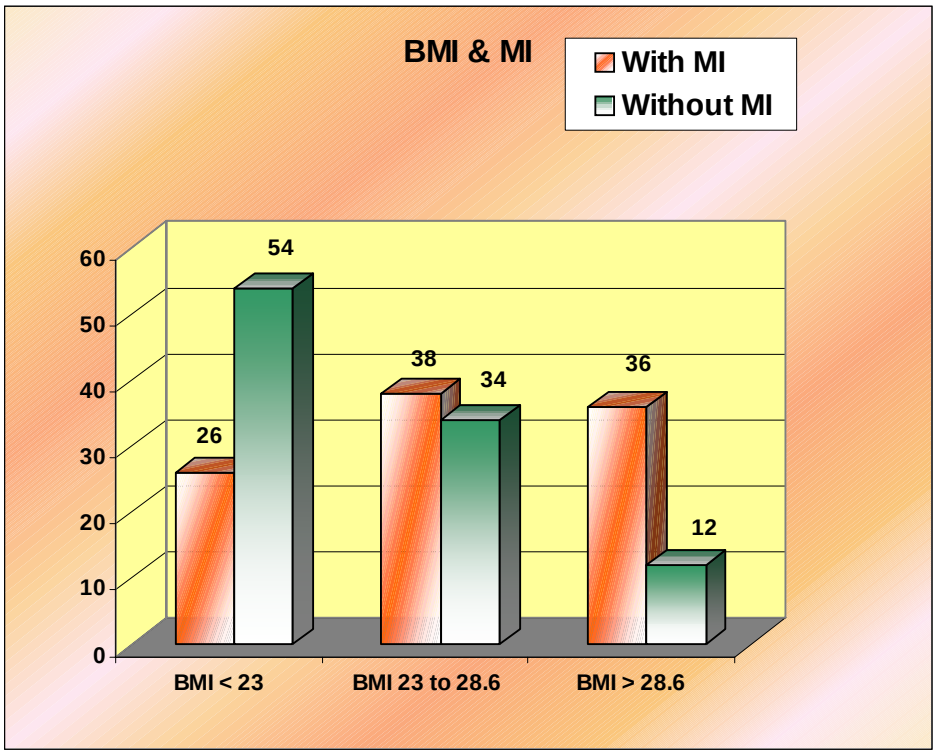


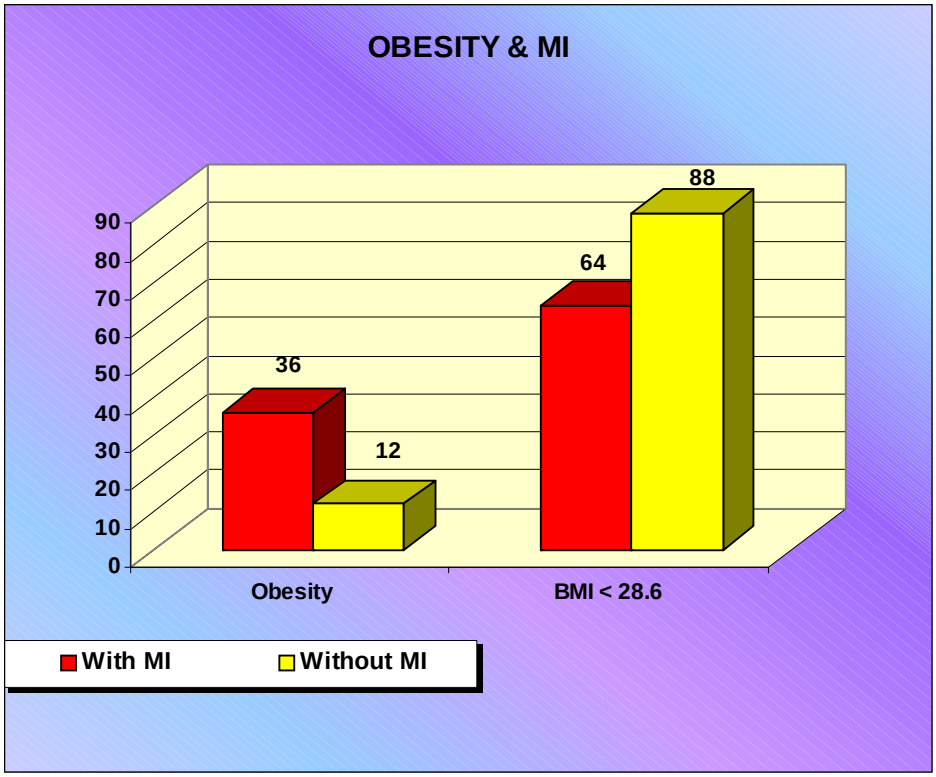
LDL > 130 mg/dL LDL<130mg/dL

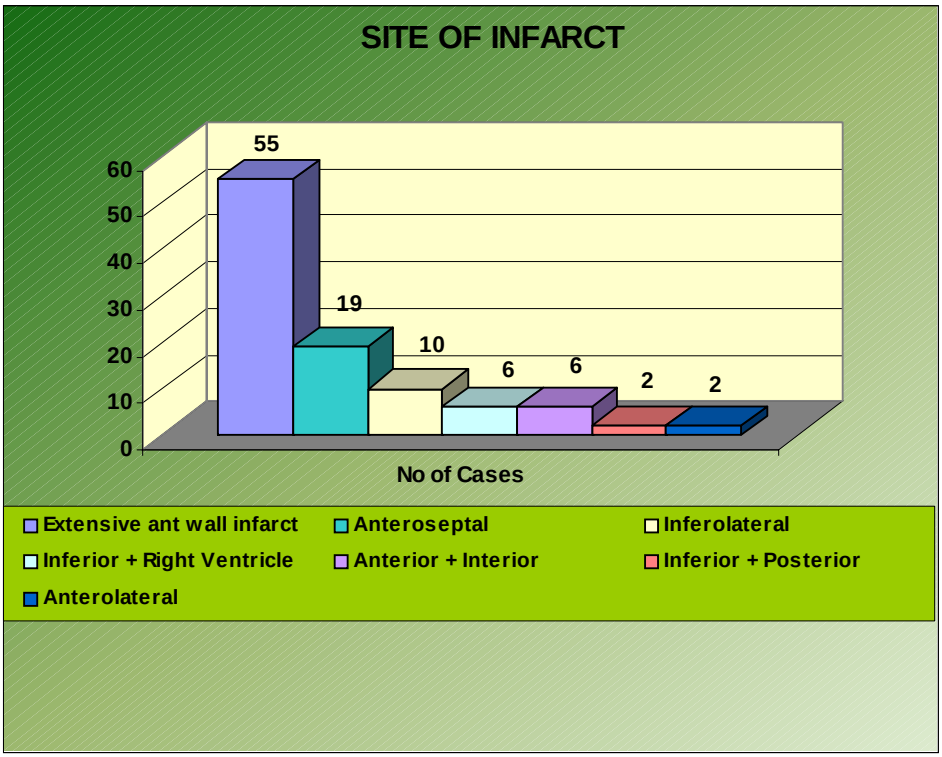
HYPERTENSION & MI

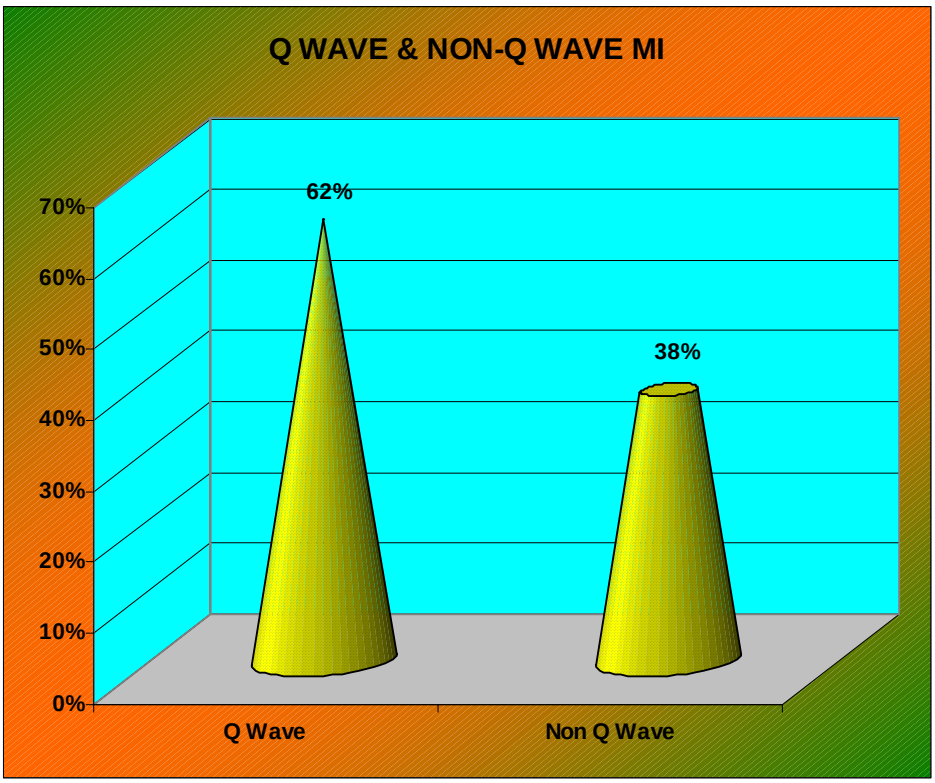


With HT Without HT


BMI & MI
With MI
Without MI
60
50
40
30
20
10
0
54
26
38
34
36
12
BMI < 23
BMI 23 to 28.6
BMI > 28.6


OBESITY & MI
90
80
70
60
50
40
30
20
10
0
36
12
64
88
Obesity
BMI < 28.6
With MI
Without MI


SITE OF INFARCT
60
50
40
30
20
10
0
55
19
10
6
6
2
2
No of Cases
Extensive ant wall infarct
Anteroseptal
Inferolateral
Inferior + Right Ventricle
Anterior + Interior
Inferior + Posterior
Anterolateral


Q WAVE & NON-Q WAVE MI
70%
60%
50%
40%
30%
20%
10%
0%
62%
38%
Q Wave
Non Q Wave

MASTER CHART

Sl. No	AGE	SEX	CLINICAL SPECTRUM - SYMPTOMS & SIGNS																		RISK FACTORS									
			CP	*RA*	*S*	*N/V*	*DW OC*	*DWC*	*F*	*P*	*EP*	*H*	*RJ*	*PE*	*AS*	*S3*	*S4*	*MR*	*R*	*W*	*SM*	*HT*	*DM*	*FH*	*OB*	*LDL*	*HDL*	*LSES*	*SL*	*PSS*
1	35	M	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	+	+	+	+	-
2	40	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	+	+	-	-
3	34	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	-
4	28	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	+	-	+
5	38	M	+	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	-	-	-	+	-
6	38	F	+	+	+	-	-	-	+	-	-	+	+	+	-	-	-	-	-	-	+	-	-	-	+	-	-	+	+	+
7	34	M	+	+	+	+	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	+	+	+	+	-	-	-	+	-
8	38	M	+	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	+
9	33	M	+	+	-	-	-	-	+	-	-	-	-	-	-	+	-	-	+	+	+	+	-	+	+	+	+	+	+	-
10	38	M	+	-	+	-	-	-	-	+	-	+	+	+	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	+
11	28	M	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
12	38	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	+	+	+
13	33	M	+	+	-	+	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	+	-	+	+	-	-	-
14	39	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	+
15	34	M	+	-	-	-	-	-	+	-	-	-	-	-	-	+	+	-	+	-	+	+	-	+	+	-	-	-	+	-
16	33	M	+	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	+
17	39	M	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	+	+	+	-
18	39	F	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	+	+	+	-	+	+	-	-	+
19	40	M	+	+	+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	-	-	+	+	-
20	30	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	+
21	27	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	-	-	+	+	+
22	37	M	+	+	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	+	-	+	+	-	-	+

23	40	M	+	-	+	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	+	-	+
24	34	M	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	-	-	-	+	-
25	33	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-	+	-	-
26	37	F	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
27	36	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	+	+	-	-
28	29	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	+	+	+	-	-
29	33	M	+	-	+	-	-	-	-	+	-	-	+	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-
30	32	M	+	+	+	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	-	-	-
31	39	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	+	-	-
32	34	M	+	+	+	-	-	-	-	-	-	+	+	+	-		-	-	-	-	-	+	-	-	+	-	+	+	-	-	-
33	35	M	+	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
34	33	M	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	-	-	+	-	-
35	39	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	+	-	+	+
36	39	M	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	+
37	27	M	+	+	+	+	-	-	-	-	-	-	-	-	+	+	-	+	+	+	-	+	+	-	+	+	+	-	+	+	+
38	38	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
39	28	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	+	-	-	-
40	39	F	+	-	+	-	-	+	-	+	-	-	-	-	-	-	-	-	+	-	+	-	-	+	-	-	-	+	-	-	-
41	37	M	+	+	-	-	-	-	+	-	-	-	-	-	-	+	+	-	-	-	+	-	-	+	-	+	+	-	-	-	-
42	38	M	+	-	+	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	+	+	-	+	+	+	+	+	+	+
43	36	M	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	-	-	-
44	37	M	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	-	+	+	+	-	-
45	38	M	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	-	-
46	39	M	+	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	-	+	-
47	38	F	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
48	38	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	+
49	38	M	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	+	+	+	+	+	+
50	39	M	+	+	-	+	-	+	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	+	-	+	+	+

51	28	M	+	+	+	+	-	+	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
52	38	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-
53	39	M	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	+	+	+	-	+	+
54	38	F	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	+	+	+
55	39	F	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
56	38	M	+	+	+	-	-	-	-	-	-	-	-	-	+	+	-	+	+	-	-	+	-	+	+	+	+	-	+	+
57	39	M	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	+	-	-
58	40	M	+	+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
59	38	M	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	+	+	-	+	+
60	39	M	+	+	-	-	-	+	-	+	-	-	-	-	-	+	+	-	+	-	+	+	-	-	+	+	+	+	+	-
61	38	F	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	+	+	+	-
62	37	M	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	+	-	-	+	-	+	+	-	-	-
63	40	M	+	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	+	-	+	+	-	-	-
64	39	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	+	+	+	+
65	38	M	+	-	+	+	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	+	-	+	-	+	+	+	-	-
66	40	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	+	-	-	-	-
67	39	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	-
68	38	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	-
69	38	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-	+	-	-
70	38	M	+	+	+	-	-	+	-	-	-	+	+	+	+	+	-	+	+	-	+	-	-	-	-	+	+	+	-	-
71	39	M	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-
72	26	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	-	+	+	-	-	-
73	29	M	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	+	+	-
74	38	M	+	+	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	-	-	+	-	+	+	+	-	-
75	39	M	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	+	-	+	-
76	37	F	+	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	+	+	+	+	+
77	40	M	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	+	+	+	-	+	+	+	-	-
78	39	M	+	+	+	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	+	-	-	+	+	+	-	+	+

79	39	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	+	+	+	+
80	37	M	-	-	+	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	+	+	-	+	+	+	-	+	+
81	36	M	+	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	+
82	31	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	-
83	34	F	+	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	+	+	-	-	-
84	39	M	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	+	+	+	+	+	+
85	40	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	+	-
86	38	M	+	-	+	+	+	-	-	-	-	-	-	-	+	+	-	+	+	-	-	-	-	-	+	+	+	+	+	-	+
87	38	M	+	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-
88	38	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	-
89	39	M	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-
90	37	F	+	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	+
91	37	M	+	+	+	+	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	+	-	-	+	+	-	-	-	+	-
92	36	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	-	-	+
93	36	M	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	+	-	-
94	37	M	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	+	-	+	+	+	-	-	+
95	40	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	+	-	-
96	28	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+	+	+	+	+	+	+
97	27	M	+	+	+	+	-	+	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	+	-	+	+	+	-	-	-
98	37	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	+	-	+	+
99	38	M	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	-
100	40	M	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	+	-	+	-	-